BRAIN.

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Original Articles.

ROTATION OF THE GREAT BRAIN.

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In the spring of 1885 I propounded, as part of a general scheme of the structure of the central nervous system, the theory, that the mammalian brain during its growth rotates upon itself.[1] The rotation produces a loop or kink, giving to the whole brain somewhat the form of a ram's horn, and bringing the part which was at first in front, on to the under side at the back. At its first formation the cerebral hemisphere of the mammalian brain is directed, like that of the reptile, straight forwards; the foramen of Monro opens into the back of its ventricle, the anterior end of the hemisphere bears the olfactory bulb. In the adult condition the foramen of Monro opens into the front of the ventricle, the olfactory apparatus is attached to the inner side of the temporo-sphenoidal lobe.

This view as to the alteration in position of the great brain was the almost necessary deduction from certain conclusions to which I had come as to the connections of the olfactory tract, for I believe that I have been able to prove a direct continuity between the olfactory tract and the anterior end of the optic thalamus, *viâ*—the fimbria, fornix, descending pillar

[1] 'The Plan of the Central Nervous System.' Cambridge: Deighton Bell & Co., 1885.

of the fornix, corpus mammillare, and bundle of Vicq d'Azyr. That the olfactory nerve is connected with the anterior end of the optic thalamus I have many reasons for believing, and the circuitous route which I have sketched out is not by any means difficult to follow. Its path is easily broken off from a hardened brain, as shown in the accompanying sketch, and seems to form an organically distinct part of the brain; and besides we have, as I shall show presently, a certain amount of embryological evidence in favour of this extensive displacement of the olfactory tract. The path here sketched out for the fibres of the olfactory tract almost necessarily presupposes

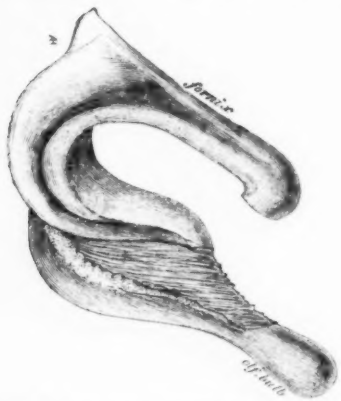

FIG. 1.—Fornix, pyriform lobe and olfactory bulb, broken off from hardened brain of Ox.

a rotation of the brain; but it is not this connection only which the theory renders intelligible. Many other features in the plan of formation of the great brain are accounted for by this supposition of its rotation. Some of this corroborative evidence I set forth in the thesis already quoted, and the object of this paper is to point out certain other peculiarities in the structure of the brain which have recently attracted my attention, as being still more favourable to the theory.

Among *external appearances*, the most suggestive is the progressive closing in of the fossa of Sylvius. The fissure of Sylvius, as is well known, is not for the greater part of its

extent in any way comparable with the other fissures of the brain. Instead of being formed like other fissures, as a narrow slit-like depression on the smooth rounded surface of the brain, it makes its appearance before any fissure, if we except the so-called rhinal fissure, as a dimpling of the mid part of the outer surface which gradually deepens into an extensive shallow fossa. The appearance of this fossa, is due, as I believe, to the traction exercised upon a part which will subsequently become the temporo-sphenoidal lobe by its attachment through the olfactory tract and bulb, to the cribriform plate of the ethmoid. As the great brain swells, its olfactory part being attached to the skull, a pitting of the outer surface is the necessary result. The fossa of Sylvius is subsequently converted into the fissure of Sylvius, a Y-shaped fissure, with a common portion leading into an anterior and a posterior limb by the outgrowth of the surface of the brain which borders it in three portions, the operculum coming down between the limbs of the Y; the frontal and temporo-sphenoidal lobes closing up beneath the operculum. The want of homology between the fissure of Sylvius and other fissures is shown, by its enclosing a considerable cortical area, the Island of Reil, which formerly formed the floor of the fossa. The radiating fissures of the Island of Reil indicate plainly enough the direction in which the traction was exerted.

When viewing the outer surface of any mammalian brain, it is easy to convince oneself that it has undergone a rotation, and that the position of its parts has been altered in such a way from the form maintained amongst fishes, amphibia, and reptiles (for special reasons it is better to exclude from the comparison the bird's brain), that the temporo-sphenoidal lobe of the mammal's brain corresponds to the anterior end of the brain of lower vertebrates.

Following closely upon the external form, the delimitation of the cortex into areas associated with peripheral nerves, presents itself as a test by which to try the theory. Recently, principally as the result of ablation experiments, the function of almost every part of the cortex has been determined, and although we are as yet far from possessing an accurate industrial map, we can nevertheless speak with assurance as to

the general distribution of work among its various territories. Undoubtedly the anterior end of the brain, particularly the Rolandic area, is in connection through the mediation of grey matter of the spinal cord with the body nerves. The localities in which sensations are received through the 8th and 5th nerves, lie somewhere in the neighbourhood of the fissure of Sylvius; the 2nd nerve has its area of cortical distribution in the occipital lobe—the 1st nerve on the inner side of the temporo-sphenoidal lobe. I have attempted to show elsewhere [1] that the observation of brains of animals remarkable either for preponderance or for deficiency of particular senses, will help us better than even direct experiment to map out the cortex into functional areas. Whatever method be adopted for obtaining the necessary data, any sketch of the brain in which functional distribution is indicated, shows clearly enough that the order from before backwards in which the areas connected with the several nerves are situated, is the reverse of that which obtains among the nerves themselves. For embryological reasons, which will be presently stated, it is only necessary to treat of the larger groups of cortical centres. The arrangement of their sub-centres, as determined, for instance, in the marginal gyrus by Horsley and Schäfer, may for the present be neglected. The body nerves have their cortical areas in front, and are followed by the nerves of the head; of these latter the optic nerve is indirectly connected with the back part of the upper surface, while the olfactory has its area in what I take to be the reflected portion. I imagine that the rotation over from before backwards is accompanied by a certain rotation from above outwards.

Passing now to the *internal structure* of the hemisphere, so far as the course of fibres within its white substance is sufficiently well known to help us, we find further evidence of rotation. Leaving out of consideration all doubtful tracts, we have the classical observations of Gratiolet, which have been frequently confirmed, to show that the anterior end of the optic thalamus is connected through the "inferior stalk" with the sphenoidal lobe, the posterior end of the thalamus through

[1] "Hunterian Lectures, 1885," reported in 'Brit. Med. Journal,' March 14, 21. Report 'British Association,' 1885, p. 1054.

the "optic radiations" with the occipital lobe. Numberless recent observations have placed the connection of the grey matter of the spinal cord with the frontal and anterior parietal lobes by means of fibres occupying the anterior part of the internal capsule beyond doubt. These three sets of fibres, anterior part of internal capsule, optic radiations, and inferior stalk, obviously cross one another, as shown in the accompanying diagram (Fig. 2), plainly indicating that the cortex of the cerebrum and the grey matter of the basal part of the system extend in contrary directions. I am afraid, however, that I can hardly render intelligible the force with which the

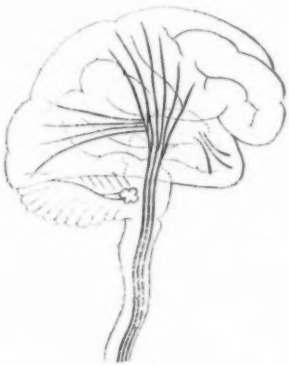

Fig. 2.

crossing of these three sets of fibres appeals to myself, without touching upon my view as to the fundamental plan of construction of the central nervous system. The system arises as an involuted tube of epiblast. Throughout the spinal region the inner wall of the tube becomes grey matter; the outer wall constitutes the white columns of fibres. In the cephalic region a second layer of grey matter, the cortex, is added on the outer side of the tube; all the grey matter of the system grows from one or other of these grey tubes. In the spinal cord, which only contains the central tube of grey matter bordering the central canal, are situated all the primary centres of the body nerves; indeed, the grey matter is entirely

used up in forming nerve-cells and plexus for the reception of sensory, and the origin of motor fibres. Travelling up into the brain, no break in the continuity of the grey matter is to be observed. It lies in the floor of the fourth ventricle, around the iter, and in the sides and floor of the third ventricle. No one doubts the continuity of plan as far as the anterior end of the iter; but hitherto it has not been recognised that the grey matter bordering the third ventricle is in functional continuity with the grey matter of the cord. The optic thalami are treated of as "basal ganglia." The olfactory and optic nerves are deprived of primary centres such as are accorded to all other sensory nerves. It is impossible for me here to point out the modifications in structure of the central system, due to the situation peripherally in the olfactory bulb and retina of some of the grey matter, which elsewhere enters into its constitution. An explanation of this will be found in the reports of my Hunterian Lectures of last year.[1] In this paper I must be content to state merely, that I regard the optic thalami as constituting the anterior extremity of the central grey tube or basal part of the central nervous system: if this position were conceded, the attachment of the extreme anterior end of the thalami with the temporo-sphenoidal lobe, of the back of the thalami with the occipital lobe, and of the spinal cord with the front of the hemisphere, would supply the strongest possible evidence of rotation.

That degeneration of a portion of the internal capsule, of the pyramid, and of the pyramidal tracts of the opposite side of the spinal cord follows extirpation of the "motor area" of the cortex, is universally recognised. The "motor area" is a congeries of local centres. The fibre-bundles which lead from local centres in the cortex to the primary motor centres in the cord, must occupy, one would suppose, discrete regions in the internal capsule and pyramidal tract. As yet considerable mystery involves their course, for it is not certain that only extirpation of the centres for the limbs and trunk induces descending degeneration of the cord. In some cases degeneration of the cord seems to have followed injury to the centres for head muscles only. It has also been observed that injury to

[1] 'Brit. Med. Journ.,' March 6, 13, and 20, 1886.

the centre for the arm may be followed by a degeneration descending lower in the cord than the primary centres for the muscles of this limb. It is very interesting to notice, however, that the only observers[1] who have turned their attention to this matter have recorded their opinion, that the anterior part of the motor area is connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the anterior (ventral) part of the lateral tract.

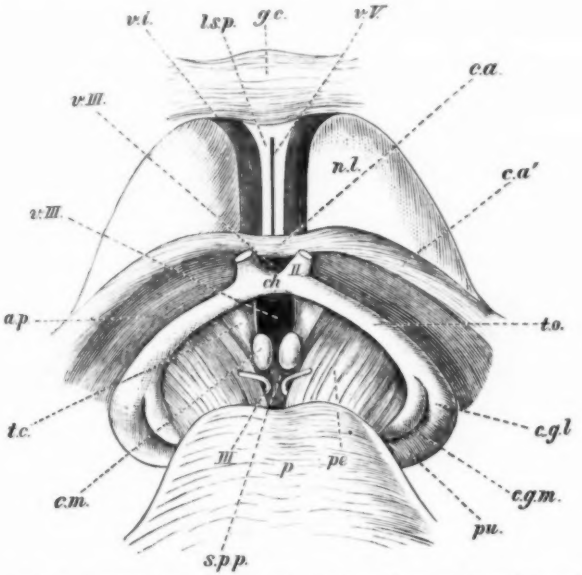

FIG. 3.—*l.s.p.* Septum pellucidum. *v.l.* Anterior horn of lateral ventricle. *n.l.* Nucleus lenticularis. *c.a.* Anterior commissure. *v.III.* Third ventricle. *g.c.* Genu corporis callosi. *c.g.m.* Corpus geniculatum mediale. *c.g.l.* Corpus geniculatum laterale. *c.m.* Corpus mammillare.

Passing now to the consideration of another intra-cerebral tract, we find in the arrangement of the fibres of the anterior commissure evidence of rotation of a most convincing kind. The accompanying illustration, which is a copy of the picture

[1] Langley and Sherrington, 'Journal of Physiology,' v. p. 49; and Langley, "Critical Digest of Recent Observations on Degeneration, and on Nerve Tracts in the Spinal Cord." 'BRAIN,' April 1886, p. 109.

on page 490 of Schwalbe's 'Neurologie,' accurately represents the peculiar twisting which the anterior commissure has undergone. It looks as if the middle of the commissure had been fixed, while its ends were twisted in such a way that the fibres which cross the median line on its under side, pass round in front to reach the upper side at its extremities. Just such a torsion, it is to be noticed, as would be produced by the rotation of the hemisphere, which I imagine has taken place, for it so happens that the rounded anterior commissure, lying just below and in front of the foramen of Monro, is exactly in the axis of rotation.

In addition to the demonstration which the twisting of the anterior commissure affords of the fact of rotation, peculiar interest attaches to it, since it enables us to fix approximately the time at which rotation takes place. It must occur after the commissure is formed. There can be no doubt that the anterior commissure appears very early in the mammalian brain, for although we have, as far as I know, no exact observations as to the time of its appearance in the individual, this much may be inferred from its great prominence in the lower forms of vertebrates. It is *par excellence* the commissure of birds and reptiles. The existence of a rudimentary corpus callosum in birds was pointed out by Meckel,[1] but the view of Stieda,[2] that no such structure exists, is endorsed by such recent observers as Rabl-Rückhard[3] and Bellonci.[4] On the other hand, Stieda recognises the existence of a corpus callosum in reptiles, but denies it in amphibia, where Leuret, Blattmann, and Reissner[5] had found it. In fishes, the commissures appear to be fused, so that no distinction between corpus callosum and anterior commissure is possible. Quite recently Osborn[6] has shown that both commissures may be found in reptiles, birds, and amphibia, although in the two former classes the corpus callosum is very ill-developed. A good

[1] Meckel, 'Deutsches Archiv für die Physiologie,' Bd. 11, 1816.

[2] Stieda, 'Zeitschrift für Wissensch. Zoologie,' Bd. 20, 1870; also Bd. 25, 1875.

[3] Rabl-Rückhard, 'Zeitsch. f. Wiss. Zool.,' Bd. 30, 1878.

[4] Bellonci, 'R. Acad. dei Lincei,' Memorie xiii., 1882.

[5] Reissner, 'Bau des Centralnervensystems der ungeschwänzten Batrachier,' Dorpat, 1861.

[6] Prof. H. F. Osborn, 'Morphol. Jahrbuch,' 1886, p. 223.

deal of the confusion in this matter has arisen, as it appears to me, from a misapprehension of the general homologies of the brain in the sub-mammalian orders. The cerebral hemisphere of the bird, and to a less extent of the reptile, is homologous, not with the entire hemisphere of the mammal, but, as pointed out by Cuvier, with little more than the corpus striatum. In the bird the cerebral cortex is very rudimentary, and consequently the corpus callosum, which is the commissure of the supra-ventricular part of the hemisphere, is ill-developed also. The anterior commissure belongs to the infra-ventricular portion of the great brain. Some confusion is due also to the complexity of the anterior commissure, which consists in mammals of two portions; (first) the pars olfactoria, which is the commissure of the inferior frontal regions (and ? the nuclei caudati), and (second) the pars temporalis. The internal olfactory tract also takes advantage of the situation of the anterior commissure to cross over to the opposite side. In sub-mammalian orders, judging from Osborn's description of Rana and other forms, the olfactory tracts reach the grey matter on the opposite side of the third ventricle independently of the commissure, which still consists of two parts, to which Osborn applies the mammalian names olfactoria and temporalis. It is open to consideration whether the homologies are not exactly reversed. For the purpose of this paper, however, we may leave out of consideration the difficulties in tracing the phylogenetic history of the anterior commissure, and content ourselves with the assurance that this commissure, in harmony with its ancestral history, is certain to develop early, and we may infer from its twisting that the mammalian brain had already reached a reptilian stage in its development at the time that rotation occurred.

The *minute structure* of the brain also affords some evidence of rotation, for the margin of the cortex mantle is for a varying distance, where it skirts the under side of the crus cerebri, invested in a sheath of grey matter quite different in structure from the cortex itself. This sheath of small-celled matter, the fascia dentata, which forms part of the cornu Ammonis, receives the thin edge of the cortex mantle. It commences beneath the uncus at the apex of the sphenoidal lobe, and extends

along the course of the posterior pillar of the fornix, reaching in some animals (particularly rodents) almost to the point where the fornix dips down in the anterior pillar. The fascia dentata is, in my opinion, identical in constitution with, and indeed a continuation backwards of, the nuclear (glomerular) layer of the olfactory bulb. In straight-brained animals the hemisphere terminates in the olfactory bulb. In twisted brains the olfactory bulb is attached to the apex of the pyriform lobe (the gyrus uncinatus of human anatomy). The continuation of its nuclear formation, as the fascia dentata, obliquely beneath, behind, and above the crus, simply shows the direction in which rotation of the great brain has taken place.

Ontogeny.—Evidence obtained from direct observation of the brain in successive stages of its growth must obviously, when the subject is thoroughly worked out, be the first to be submitted in proof of the rotation of the hemisphere. On account, however, of its scanty nature as yet, I have reserved it until the end of this paper. Partly its deficiency is due to the absence of research, partly it depends upon the peculiar nature of the organ itself. Unlike other tissues of the body, the nervous tissue is peculiarly plastic. In the course of its growth it may receive marks of torsion and other displacements, but while in other tissues such marks permanently remain, in the brain they are obliterated by further growth. The manner in which its mass is added to also has an important bearing upon its form. It must be remembered that only the cellular elements enter into its original constitution; the fibres are but processes of the cells. Of the primitive epiblastic cells involuted to form the nervous tube, some, in all probability, are favoured above the rest and become the functional nervous elements, while to the others only subsidiary, supporting, nutritive and insulating functions are assigned; they become the neuroglia and myelin cells; just as in the case of each little group of epithelial cells involuted into a follicle of the ovary, one only becomes an ovum, the others minister to its wants. Most likely, therefore, the lines along which nerve fibres are to grow are in many cases laid down, before the fibre itself appears, by the chain of accessory cells which have taken

up their position along its route, but it is open to us to suppose that, in other cases, the route chosen by a fibre depends upon convenience at the time of its appearance. If, owing to a displacement of the cortex, it can reach its destination by a shorter route than the tract occupied by neighbouring fibres, which being fully developed at the time of the rotation of the brain, shared in the displacement of the cortex, it is open to it to leave the original tract, and thus the primitive arrangement of the constituent parts of the brain may be masked. Despite, however, the obliteration of the indications of rotation which these two causes are likely to produce, we still can see, I think, in the changes of form which the great brain undergoes signs sufficient to prove that rotation does occur. Undoubtedly this is the conclusion one is likely to draw from the examination of three such pictures as are here inserted:

Fig. 4.

Fig. 5.

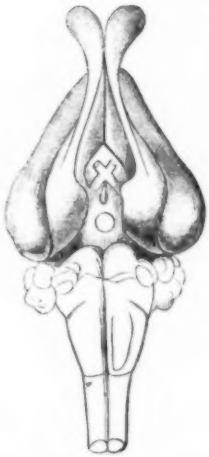

Fig. 6.—Ventral surface of brain of fœtal rabbit, after Löwe.

In Fig. 4, copied from Löwe,[1] a very early stage in the growth of the brain is shown, the hemisphere is directed forwards and bears the olfactory bulb at its anterior end.

[1] Löwe, 'Entwickelungs-geschichte des Nervensystems,' Pl. I., figs. 1 and 2.

Fig. 5 represents the appearance presented by almost any mammalian brain after the formation of the rhinal fissure. The olfactory bulb is now attached to the extremity of the pyriform lobe, and the suggestion of a folding over of the whole hemisphere is very strong. The pitting of the side of the brain due to the tension on its posterior end produced by the attachment of the olfactory bulb to the cribriform plate, is beginning to appear as the fossa of Sylvius. Fig. 6, again, after Löwe, is inserted for the purpose of showing the continuity of the olfactory bulb, pyriform lobe, and gyrus uncinatus. It is curious to notice how the backward growth of the hemisphere increases the size of the loop. In the brain of a sheep, for instance, the fimbria (posterior pillar of the fornix) is transverse at its first appearance. In marsupial brains it never becomes more than very slightly oblique. The higher the animal in the mammalian scale, the more nearly does its fornix system assume a longitudinal direction.

Of the earliest stages of the growth of the brain we have very few observations, but Marshall's[1] discovery that the olfactory nerve arises, like all other sensory nerves, from the neural ridge, and that it is then pushed down in front by the outgrowth of the cerebral hemispheres, to be again caught up in the cortex mantle, opens up great possibilities with regard to the relation of the olfactory apparatus to the hemisphere, and prepares us, although the research has gone no further, to find an arrangement in the first nerve quite unlike that of the others.

Phylogeny.—The reference already made to the marsupial brain has drawn attention to the fact, that in the lower mammals the rotation has gone less far than it has in the higher members of the class. Whether or not it is a phase in the development of any sub-mammalian animals, I am not prepared to state. Certain it is that, if we descend as far as the reptiles, rotation is out of the question. As already pointed out at the commencement of this paper, the reptilian brain, like the brain of the early mammalian embryo, is directed forwards, its ventricle communicating with the third ventricle behind through the foramen of Monro, and ending in the

[1] Marshall, 'Quarterly Journal of Microscopical Science,' July 1879.

ventricle of the olfactory bulb in front. The great gap between the bird's and mammal's brains, although bridged over by the brain of the monotreme, renders it unlikely that the link between the straight and the rotated brain will ever be found. The peculiar deficiency of cortex in the bird's brain renders rotation unnecessary, for I do not think that the *reason for rotation* is far to seek. If we compare such an extreme type as the crocodile with any mammal, we notice a contrast in the whole architecture of the head. The reptilian head is directed forwards; is elongated in the direction of the axis of the body. The mammalian head approaches a globular form. With the greatly increased size of the brain as we ascend in the vertebrate scale, economy of space for its reception becomes necessary. Doubtless, for other reasons also, a shortening of the head is desirable, and the larger brain can only be packed inside the shorter skull by throwing its hemispheres over into loops.

SOME OF THE RELATIONSHIPS BETWEEN EPILEPSY AND INSANITY.

(Read before the Neurological Society of London.)

BY C. H. SAVAGE, M.D., F.R.C.P.

Though I have no great practical experience with epileptics as a class, yet, having so much to do with the insane and with their relations, I have had opportunities of seeing at least some of the relationships which exist between the two classes, and therefore, in deference to the wishes of Dr. Hughlings-Jackson, I shall give you the result of this experience. I shall not give any definition of epilepsy, for I feel that at present we have to look upon it as a "disease" provisionally, and that the time is at hand when it will be looked upon only as a group of symptoms which happen to occur together, and which will be seen to occur together under many varying conditions.

I would only say, for convenience, that I make clinically two main divisions among epileptics, namely, the neurotic and the accidental, the functional and the organic, if I may use these terms without connoting too much. I mean to imply, that there are many cases in which the epilepsy seems to me to belong to a state of inherited nervous instability; but beside these, there are many other cases in which, sooner or later, we shall learn that there are definite lesions in the brain itself, giving rise to the epileptic symptoms. The first set of patients belong to the insane class; the second, on the other hand, need have no such connection, but are allied to those cases which follow injury or coarse brain changes.

But returning to the consideration of the relationships between the two disorders—and by relations I wish to imply not only likeness but contrast. Epilepsy and insanity are alike in origin. Similar causes will produce insanity and epilepsy. Probably both are often the result of one cause, and we must never forget this in looking at the relationships between these disordered states.

As insanity is very often the direct product of insane parentage,

so epilepsy may result from epileptic parentage. But further, there is a family peculiarity which we call neurotic—from neurotic parentage we often see both insane and epileptic offspring. By neurotic parentage I mean that there are certain degenerate families in which we meet great nervous instability, a tendency to show bodily weakness by nervous perturbation. This nervous instability may be shown either in loss of control or loss of power, and this loss may affect either the motor or the mental side chiefly.

But to further develop this fact, that epilepsy and insanity are allied by origin, I must give some facts as examples.

We often meet with both insanity and epilepsy in the same family; we may meet with epilepsy in a parent and insanity in the children, or we may meet with insanity in the parent and epilepsy in the children.

In my experience it is very common to see the epilepsy in the collateral branches of the insane family, but we must not forget the important fact, that there are more members of the collateral branches than of the main stem.

Besides neurotic inheritance, I believe that there are some special causes of insanity which are more likely to produce epilepsy in the offspring. Thus I believe that insanity produced by alcohol, or by coarse brain injury, is very likely to re-appear as epilepsy in the next generation. I do not give this as a dogmatic faith. But I have seen it occur myself, and I find that writers on alcoholic-insanity give many cases in which this connection is manifested.

I think there is a difference then between the children of certain insane persons and those of certain others, suffering from insanity, and I would put it thus; that whereas it is common to meet with the children of the truly neurotic parent suffering from idiocy, moral insanity or moral obliquity, or from the more chronic and organised forms of insanity with hallucinations, such as those cases described as delirium of persecution or paranoia, it is more common to meet with epilepsy in the offspring of those who have become insane from some local degeneration or disease of the brain. I trust this subject will be studied and supported, or refuted, according to the truth that is in it.

Epilepsy may occur then in the same family as insanity, being produced by similar predisposing and exciting causes. And there are some other general conditions which set up both the one and the other in families which have been free from any taint of neurosis, and this too is a fine field for the investigator. I subjoin some instances.

The eldest daughter of an eccentric but highly gifted man died of acute delirious mania, which followed an attack of measles; another daughter suffered from chronic insanity, with delusions of a very fixed nature; while the son suffered for some years from epilepsy.

In one other a still more interesting relationship existed, for of twin children, one suffered from insanity and the other from epilepsy.

In other cases, idiocy and insanity, and epilepsy and idiocy, have presented themselves in different members of the same family.

So much then for the relationships of origin, and I need not repeat what I have so briefly put before you.

I now pass from the development of epilepsy in the same family to the occurrence of both epilepsy and insanity in the same individual.

Epilepsy may be preceded by some mental disorder, and this disorder may vary in degree from the simplest hallucination of a sense, occurring as an aura, to a furious outburst of complex but uncontrolled actions. Hallucinations of any of the senses may occur, and though I have frequently met with hallucinations of smell, I would not like to say more, than that they occur in my experience more frequently with epileptics than with those suffering from insanity. We meet with hallucinations of sight, and these may assume the character of an explosion, if I may use that term; they may appear as flashes or sudden rushings of light; the hallucinations of sound, too, may be explosions or "voices," and though I do not attribute much importance to the experience, I think that what appear to be directing voices do occur sometimes before as well as after the fit. The hallucinations preceding the fit may colour the acts performed after the motor disturbance has passed off, and a threatening voice as an aura may be the cause of a violent attack, which is performed during the post-epileptic automatic state.

As a rule, I believe these auras or these delusions are, like the rest of the fit, regular and constant in their origin and their course. It is quite impossible to separate the periods clearly, for if very slight fits occur, the actions which appear to replace the fit really follow it.

In the following case there seems no doubt the symptoms—mental—are mixed up with the fit. The case is known to your chairman—Dr. Wilks.

A busy provincial lawyer, especially when over-anxious, as at election times, had what he called a very strange experience. Tired, he would go to bed and sleep fairly well, but he would have a very painful dream. He would feel that he was brushing his

teeth rather violently, and that suddenly the tooth-brush would slip from his hands, and then that he was choking. He would struggle for his breath, and during this struggle he would lose himself, and only remembered the dream in the morning, when he woke, dull and tired, to find a spot of blood on his pillow. As I said above, this dream repeated itself in every particular, like other true epileptic seizures.

It seems to me that the step between this man and the somnambulist is very short; the unconsciousness of the latter makes the difference. I cannot help thinking that a very little more stimulus would have made the lawyer do something, in his struggle for breath. Sense perversions, or more organised insanity, then, may precede epileptic fits.

In certain cases insanity is supposed to take the place of the fits altogether, and it is not necessary for me to go into all the evidence which has been brought forward to support the existence of "L'épilepsie larvée;" my own opinion, supported by Dr. Hughlings-Jackson and others, is that in most of these cases a fit of some sort, great or small, does occur before the strange acts. This marked epilepsy is less common in England, or at least has been less commonly studied in England, than in France, and it is interesting that France should produce such peculiar varieties of neuroses; there seems to me to be a specially nervous element for good and ill in that nation. I have met with but few cases of true masked epilepsy, and in none have I been convinced that no fit had occurred. The acts similar to those described by French authors are not so very rare, and I would give a case which I look upon as a good example of the kind of fit which is supposed to be masked.

A young labouring man, belonging to a nervous family, that is, to a family where neuralgia and mental peculiarity occurred, took to drink, developed epilepsy, and later became insane. From the epilepsy developed violent outbreaks of mania, and for these he was sent to one of the county asylums. After treatment, he recovered from his insanity, and had almost got rid of his fits, these only coming on at distant intervals. As they were always preceded by a distinct aura, he was able to manage himself very well; he continued at work and was steady. There were, however, occurrences for which he could not quite account, and there were lapses in his life he could not fill.

The mystery was cleared up in this way. As he was walking along a country lane he felt his fit coming, and loosened his collar and necktie, as he had learned to do in the asylum. He got to the roadside and lay down; on recovering his senses he could not

understand the nature of things, for he found himself at the distance of some miles from where he loosened his collar. He heard later that he had been seen and met by some who knew him, and that he passed them without appearing to recognise them. His gait, too, was said to have been staggering, so that his neighbours not unnaturally thought that once more he had taken to drink. Doubtless the history was this, that after the aura came the fit; then there followed the slight rest, followed by an automatic state, in which the journey was pursued, till a period of exhaustion set in, associated with sleep, and the awakening took place at a distance from the site of the fit.

Later I shall show how this case connects the epilepsy with epileptic fury.

Epilepsy may not only be preceded and replaced by insanity, but very commonly indeed it is followed by mental disorder. We shall in this great class find hundreds of patients who fill the asylums for chronic lunatics.

Epilepsy seems to have a very destructive influence on the mind, and here it will be well to study some points in relationship to the mental weakness following epilepsy.

Is the weakness the result of the exhaustion produced by the severity of the epileptic discharge, or is the destruction due to mental instability (inability to construct and repair), resulting from the disturbance due to fits; or do these epileptic attacks set up a process which does not end with the fit?

There is one other consideration which I think deserves more careful study. It is often said we have too many cases described as epileptic, as though there were only one set of mental relations to one set of epileptic ones.

I believe that study will show, that among the hundreds of epileptic lunatics there are many sub-groups quite worthy of names. There are certain cases in which the convulsions are associated with certain sets of mental perversions, and there are certain other cases in which the fits take other relationships to the mental disorder. This field is a large one, and one at present but badly tilled.

I am constantly talking of "functional" disturbance; and though I know this is rather heterodox, when we are supposed to find a physical basis for everything which occurs, yet I for one am forced to see cases in which I cannot help blaming the function before I do the organ. And in studying epilepsy I wish to say that I believe there may be functional equivalents to general paralysis of the insane, and just as we have decay of power associated with loss of control, just as we so commonly have fits

towards the end of general paralysis, so I believe among the chronic epileptics of the asylums will be found patients who are perverted functionally, and with recurrent discharges of an epileptic nature. This part of my paper is highly theoretical, and is very open to debate. I merely suggest the possibility, and I shall be glad to hear more from others.

There is no doubt that insanity of the weak-minded type follows the more *frequent* rather than the severe fits. I should like to hear whether it is true of epilepsy as of insanity, that those who are epileptic by origin can bear much more nervous disturbance than can those who are apparently of good stock. The more unstable are more easily upset, but again more easily reset. It is true that, in some cases to be referred to again, a single fit is enough to upset the mind and render it weak for life, but this is not the rule; the constant repetition of fits is supposed, by some, to be like constant spirit-drinking, a cause of constantly recurring hyperæmia which tends to degenerative changes.

It is self-evident that constantly recurring disturbance of function will lead to the injury and destruction of the organ, and I do not believe, notwithstanding all that has been said about the special circulation in the brain, that disturbance of the motor areas constantly recurring, will leave the rest of the brain unaffected by the neighbouring change.

The fact remains that there are a very large number of chronic epileptics who are weak-minded. Also, I believe the study of these cases will be as useful to the physiologist and to the philosopher, as is the study of the weathering of the rocks to the geologist.

There are almost certainly among these weak-minded epileptics at least several groups in which the epilepsy is but a small part of the disordered process.

We have to learn that all fits are not alike, or always associated with a special sort of mental disorder, and, what is more, I shall have to point out that fits may not always be the worst part of the diseased action. The relation of the fits to the age of the patient is important. We all recognise that constantly recurring fits produce weakness in mind, and we also see that the younger the age when the fits appear, the worse is the prognosis, so that fits in infancy often prevent all further mental development.

Disturbed nervous conditions and epilepsy are not uncommonly associated with adolescence, and here we have to do with a more stable age than childhood, yet one eminently unfit for severe shocks, and likely to suffer greatly in mind by fits. As manhood advances, the onset of fits has a graver meaning, but then only as

probably being associated with other and more tangible changes in the brain itself. And when age comes on, the same thing holds of age as of childhood, the power of the fit for evil seems very great; in the one case, the edifice is not yet built and founded firmly; and, in the other, it is tottering to its fall.

So far, I have inverted the usual order by beginning with the study of the more chronic mental changes produced by epileptic fits, before examining the more acute symptoms which may follow directly on the fit. I have now something to say about the acute mania which frequently follows an epileptic fit.

The fit may be severe or not; the patient may sleep for a longer or shorter time, and may suddenly or gradually become excited. He may perform the most complex actions, and may speak and appear to hear what is said to him, and yet in most of these cases he will have no recollection on his recovery of anything which he has done. As I said before, the state is a condition allied to that which is called automatic, and his acts are in many cases a direct result of some sensory or higher intellectual discharge. If I may use the term, the machine seems to be set in motion by some sensory or other disturbance, and works along the usual track as if it were a mere calculating machine. I know in using the term "discharge" I shall lay myself open to criticism, on the ground that there cannot be any, or at least many, intellectual centres answering in any way to motor centres; but I can best explain what I mean, by referring to other conditions in which, the higher control being removed, there is a similar discharge, that is, an unrestrained running away of force along certain lines.

Thus, on the one hand, we meet with monkey-like idiots who are never still for a moment; they are never saving or storing any force for future use; the force seems to be expended in any reflex way as soon as it is made, and we see a very similar state of things in the earlier stages of dementia in some elderly persons.

The higher control removed in both these cases is seen in the unrestrained exhibition of energy, and, as a rule, it is mischievous and destructive, and with but little memory of what had occurred. I do not wish to push the likeness too far, but I only use the comparison to explain what I mean by intellectual discharge.

Epilepsy may be followed at once by fury, but, as a rule, there is a period of rest between the fit and the fury.

The mental disorder may be senseless destructive violence without any apparent aim or object in view, or it may have the aspect of design. In this latter case it may depend on the delusion or the sensory perversion which started with the aura, or it may result as a kind of intellectual reflex due to some sense impression

arising during the fit or immediately after it. The actions may point to some fear of being followed or persecuted, and may be perfectly consistent with ideas of self-defence. There is nothing in any way irrational in the acts themselves in many cases, though in others these acts are incoherent, if I may use such a term, that is, they are "mixed," part of one set of actions being confused with another set. He would be very rash who would pretend to dogmatise and qualify the nature of what acts may or may not be done in an unconscious epileptic state.

The duration of the maniacal excitement is another point of great interest to me, for it seems as if there may be a mental side to the status epilepticus, just as there is a motor one. In post-epileptic mental disorder it seems as if a process were often started which is very hard to reverse or stop.

The starting of epileptic mania by a sudden muscular disturbance recalls to my mind those cases of insanity in which some acute cause—such as delirium due to fever, alcohol, belladonna, or even to chloroform—has sufficed to start a maniacal process, which has continued for weeks or months. One other point of interest is that there is a likeness between the two diseases, epilepsy and insanity, from the treatment, bromide of potash having as high a reputation for relieving the one as the other. I do not for a moment wish to disparage the use of bromide in both disorders, but it would be unreasonable to suppose that a disorder, which I believe to be but part of many morbid processes, and arising in many varying conditions, is to be treated always by one remedy because one of the chief symptoms happens to be fits.

I hear from some asylum superintendents that they do not give bromide at all now, as they find that the patients do not seem to do so well when these fits are entirely suppressed; and I should, from my point of view, say that that asylum is not the best where the patients are the most quiet, if the quiet be the result of drugs. That I have ground for believing that harm may follow bromide, I will show by giving one example from many.

A girl of weak intellect from childhood developed fits, occurring at long intervals, mostly at night; these never attracting much notice till she approached maturity, then they were vigorously treated, and the child became much worse in mind, so that she was now almost always restless, excitable, destructive, and dirty. The mother's own words were, "Doctor, there are worse things than fits." She then told me that when the child had the fits she was always brighter and more companionable for weeks after, and only unruly just before their onset. This case is not singular. I could record several others, but one suffices for my object.

Another relation calls for attention.

Either epilepsy or insanity may be replaced by other neuroses. I know that epilepsy may be replaced by migraine or asthmatic attacks, and I have seen several good examples myself in which insanity has been replaced by similar disorders.

I have incidentally said that there are worse things than epilepsy, and I should not feel that I had completed my paper without referring to cases in which epilepsy seemed really to have had a chief part in restoring the mental balance. First of all, my attention was called by several cases of rather chronic insanity, in which severe fits were followed by recovery. In one, a case of profound melancholia, a series of very severe fits in which there was sub-conjunctival haemorrhage and numerous bloody puncta under the skin, was followed by steady improvement and ultimate recovery.

In a second, the stupor following an acute attack of insanity seemed likely to pass into permanent dementia, when fits occurred, the patient remaining in a status epilepticus for many hours. From that time she steadily improved, and was discharged well. In the next place, I have met with several cases of general paralysis of the insane in which mental clearing up followed fits. This is the exception; but these cases were clear, and without any other recognisable cause for improvement. I might here say that this point is also made more interesting from the fact that, in some cases of general paralysis of the insane, a bodily illness has once or twice been followed by recession of the acute mental symptoms.

The most striking instance I have to give of mental improvement after fits is the following. A child of a very eccentric father and with neurotic sisters, was so weak in mind, that his education was all but given up. He was never expected to be more than an imbecile, and about fourteen was utterly uneducated. Then for the first time he had fits of an epileptic nature; and from that time he developed, first slowly, and then more rapidly in mental and bodily power, till at eighteen he had attained a good education and lost his fits. He gained an open scholarship, and is now a promising student at one of the Universities.

It is well to remember when speaking of fits, that though there is a common occurrence of fits in general paralysis, yet the two—epilepsy and general paralysis—are very widely separated; and it is strange that, among the many epileptics in a county asylum, very few indeed, if any, die of general paralysis of the insane. I feel I have taken too much space, as it is, with my rather wide wanderings, but I cannot conclude without putting down a few jottings by the way. I think if it be a fact that hallucinations of smell are more

common than other hallucinations in epilepsy, some explanation is needed. Hallucinations of smell occur in the insane frequently with sexual disorder, but I see no such relation between epilepsy and sexual perversion.

I think we should study the rate and direction in which destruction of mind due to epilepsy takes. I believe memory is very early and very gravely affected, but that loss of control is the chief effect in the furious cases; so that here we have two groups, one of those tending to dementia, the other those to mania as results of mental losses in different directions. Though impulsive violence is frequently a characteristic of epilepsy, it is not safe to say of every impulsive act done by a lunatic or by an unstable person, that it is epileptic, unless there be a history of fits or of a strongly neurotic family history.

Again; though one chief characteristic of epilepsy is the exact repetition of the symptoms, it does not follow that exact repetition of symptoms connotes epilepsy; as most of us in asylums have met with cases in which the same ideas, following in the same order, give rise to the same delusions and the same insane acts in attack after attack of insanity.

Before winding up, I have a few words to say on general relationships of epilepsy and insanity. Writers have long spoken of an insane aspect, and others of an epileptic facies. There is some truth in both, but not very much, as epilepsy may last for years without leaving any special impress on the features. It has been said that the chronic epileptics assume peculiar mental aspects, and that there are two well-marked groups of these; the first containing the more dangerous class—those who are suspicious and irascible—the other, those who are mild, sheepish. These are, to my thinking, the results of the partial weakness of mind; the one being a state of nervous irritability, easily roused, while the other is weakness and unimpressionability. Epilepsy has for ages been credited with alliance with greatness, and we all know of the great men who have been reputed as epileptics. I would first of all say, that there is little evidence that any of these were really chronic epileptics, the truth being that fits of some sort were present at some period of their lives, and these were all called by one name. It is interesting to find that here, again, we find the alliance between epilepsy and insanity; just as some genius is near insanity, so some fits are met with also in some intellectually great men.

I fear my task now done is but feebly performed; but if it lead to the study and development of these relations and others, I feel sure light will arise, the brightness of which we cannot guess.

ON PSEUDO-TABES FROM ARSENICAL POISONING, WITH A CONSIDERATION OF THE PATHOLOGY OF ARSENICAL PARALYSIS.[1]

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THE two cases which I here present, illustrate the fact that arsenic may cause very typical "pseudo-tabes."[2] The cases also, when studied in connection with recent observations regarding alcoholic, diphtheritic, and plumbic ataxia, make it almost certain that arsenical paralyses are mostly of peripheral origin.

CASE I.

A. C., age 48; single, American.

His mother had a chronic gastralgia of many years' duration. When 20 years old he began to suffer from the same complaint, and as a result became a morphine-taker.

Having reached the point where the amount of morphine necessary to control his pain made him utterly unfit for work, he came to my ward in Bellevue Hospital. At this time, Dec. 21st, 1885, a physical examination showed no organic disease.

Knee-jerk slightly exaggerated, other reflexes normal; urine normal.

On Jan. 1st he was placed on small doses of Fowler's Solution for the relief of his gastralgia, which returned whenever the morphine was withdrawn. The dose was gradually increased until during the whole of April he took ozss. t. i. d. In the latter part of this month the patient developed symptoms of motor and sensory irritation and paralysis involving all the extremities.

There was first a burning feeling in the fingers, toes, and soles of

[1] Read at the Annual Meeting of The American Neurological Association, July 21st, 1886.

[2] The term "pseudo-tabes," has been criticised, and with some justice; but it has acquired a wide use and a pretty definite significance in neurology, and so I have employed it.

the feet and an itching of the eyes; then a gradual weakness in the limbs developed, with uncertainty in gait and clumsiness and inability to use the hands. He had to give up writing. The burning feelings were followed and supplemented by sensations of numbness and prickling, and occasional sharp pains in the legs. No vesical or rectal trouble was observed. Examination at this time showed the patient to have an uncertain irregular gait. The ordinary tests revealed both static and locomotor ataxia with diminution of muscular sense. He could not stand or walk with his eyes closed, or touch the tip of the nose with the fore-finger, &c. &c. The patellar tendon-reflex was abolished in the right leg; almost so in the left.

A patch of anæsthesia existed on the dorsum of the right foot between the first and second toes in the distribution of the *peroneus profundus*. The patient felt as though walking on wool. There was muscular tenderness in legs, especially over motor points. Optic neuritis was observed on ophthalmoscopic examination, kindly made by my friend Dr. Leszynsky. The pupillary reflexes were normal. There were some tremors and muscular twitchings. There was great motor weakness in all four extremities. Dynamometer: right hand 22; left hand 30. The electrical reactions could not be taken until the patient was very much improved. They were then found to be, for the arm muscles, about normal.

For the lower limbs:—

Faradic current: Electrical sensibility greatly diminished.

Electrical contractility, very much diminished.

Galvanic current: Electrical sensibility greatly diminished.

Electrical contractility normal.

Urine normal.

After discontinuing the arsenic the patient slowly improved, but it was five months before all parasthesiæ were gone.

Case II.

The next case was a more striking one. The patient, Jos. B., 29, single, German, varnisher, was kindly sent to my clinic at the N.Y. Post Graduate School by Dr. Wm. M. Leszynsky, whence he was transferred to the hospital.

The patient's parents were both phthisical. Patient himself had had erysipelas nine years ago, tertian ague four years ago, gonorrhœa two years ago. Never had syphilis, was a moderate drinker; not a smoker. Had been in vigorous health up to the evening of March 1st, 1886, when he got drunk and took a handful of Paris green. He vomited all night, and early in

the morning he was taken to a hospital, where his stomach was washed out.

For five days he was prostrated and too weak to sit up, but he was not paralysed. About the sixth day he began to feel the sensory troubles, viz. frightful burning pains in his extremities, formication, and occasionally cramps in the legs. Despite this he got up on the sixth day, being then weak, but having no paralysis. About this time he began to develop an ataxia, which he noticed first when he was washing himself. He even found that he tottered and was uncertain of himself when his eyes were closed. The epidermis at this time began to peel from his feet and hands. He had no bladder trouble.

On the 14th day he left the hospital. His strength was increased, but the parasthesiæ and ataxia were unchanged or worse.

He was first seen by me on April 1st, a month after his poisoning.

Examination then revealed the following: General physiognomy: a strongly-built muscular man. His gait is slow and careful. He has to watch the floor, and he brings his feet down hard, the heels striking first. His gait is characteristic of locomotor ataxia.

Mental sphere: his mind is clear, though he is nervous and worried about himself, and he sleeps poorly.

Motor sphere: no absolute paralysis, but much muscular weakness, and he cannot walk up or downstairs, or raise himself on a chair. The paresis is most marked in the extensor cruris of both extremities.

There are no tremors or spasmodic movements.

Dynamometer: right hand 22; left hand 22.

There is great tenderness over the course of the sciatic nerves and over the branches of the brachial plexus in the arm and fore-arm.

Sensory sphere: there is cutaneous hyperalgesia of all four extremities.

Tactile sense, normal; temperature sense, normal. Muscular sense: the patient shows an almost complete loss of muscular sense with consequent ataxia in arms and legs. He cannot tell the position of his limbs, or distinguish between weights differing 1 to 40.

Reflexes: the patellar tendon-reactions are abolished. There is no ankle clonus. Plantar and cremasteric and other skin reflexes are present and normal.

Eye: pupillary reflexes all are normal. Ophthalmoscopic examination by Dr. Leszynsky showed the fundus to be normal; but patient complained of impaired vision.

Mechanical irritations (blows) over motor-points produce no contractions, but striking the body of muscles (t. anticus) produces "muscle waves." On the chest myoid tumors can be raised.

Trophic :— The epidermis has peeled from the feet and hands, once, completely, and a second time partially.

There is some atrophy of the arms and legs.

There are no secretory disturbances. The bowels are regular.

Urine :—At first on admission, sp. g. 1010 acid; clear; no albumen.

One month later (two months after poisoning), sp. gr. 1012 acid; straw colour; trace of albumin (from a gleet?).

Electrical reactions :—

Muscles of Legs.

Far. current.—No contraction.
E. sensibility lessened.
Galv. current.—ACC almost = KCC.
AOC < KOC.
Contractions sluggish.
E. sensibility lessened.

Thus there was shown to be a partial RD.

Muscles of upper extremities, normal.

The patient improved, but very slowly, the burning sensations giving him most trouble.

He became slightly hysterical in his anxiety over himself.

Cases of the character that I have described are not unknown in literature. Possibly they are not so very rare. The brief descriptions of older writers often indicate that the paralysis approached this pseudo-tabetic form.

Yeesche (*loc. cit.*) reports a case of acute arsenical poisoning, followed a week later by cramps and neuralgic pains in the extremities, paralysis of muscular sense, tactile and pathic hyperæsthesia.

Scolusoboff (*loc. cit.*) notes a case in which there was general atrophy, some paresis, muscular pains and tenderness, loss of muscular sense and hyperalgesia.

Dupuy (*loc. cit.*) reports briefly the case of a patient who, after taking arsenical baths, suffered from parasthesia and diminished sensation in the extremities, "with uncertain gait."

Levin (*loc. cit.*) reports the case of a man, aged 17, who was using an arsenical salve. He suffered from a condition of

nervous prostration, followed in 4 or 5 weeks by complete ataxia of all extremities, and a gait which resembled a high degree of chorea.

Seeligmüller (*loc. cit.*) reports the case of a young man who was poisoned by arsenical wall-paper. His symptoms resembled so closely those of locomotor ataxia, that he was declared at first to have that disease. Later the true nature of the trouble was made out. It was distinguished from true posterior spinal sclerosis, by the presence of tendon-reflexes, burning pains in extremities, weakness of extremities, absence of girdle symptoms or vesical trouble.

As to the pathology of arsenical poisoning, authorities differ. In the papers of Da Costa, Mills, and Seguin, published four and five years ago, the view was held that the anatomical change was a diffuse myelitis.

Da Costa thought it more of an anterior poliomyelitis. Others have seen in it a resemblance to the subacute spinal paralysis of Duchenne. German authorities (Strümpell, Leyden) have rather inclined to the view that the disease is a neuritis.

No autopsies have yet been recorded. The only reference to such is that of Wibmer (*loc. cit.*), who says that the lower part of the cord is always affected, as is shown by the frequent congestion of the cauda equina.

Apart from the clinical history, the most conclusive evidence of a myelitic origin was thought by Seguin to be the experiments of Popow, published in 1881, upon rabbits.

Kreyssig, a year ago, however, working under Schultze of Heidelberg, found in the spinal cords of healthy rabbits all the changes which Popow described as being present after arsenical poisoning. On the other hand, in six rabbits poisoned with arsenic, he found no pathological changes in the cord.

Pistorius states that the nervous system of rabbits and guinea-pigs is very sensitive to arsenic, while that of cats and dogs is less so. It is very evident, therefore, that no pertinent conclusions could be drawn from experiments with rabbits, even if there were any such data to start with, as Popow believed that he had. Jaeschke gave a dog arsenic in large

doses for ten days, then gave a fatal dose. Paralytic symptoms, though not well marked, developed. On post-mortem he found a few small apoplexies in the spinal meninges, but nothing to indicate a beginning myelitis.

At present, therefore, we must rely upon the clinical history of the cases to determine the pathology. But the evidence, it seems to me, here is so strong as to amount almost to a demonstration. One cannot read over the histories of cases of arsenical paralysis without observing how closely they tally with those of multiple neuritis, as described in systematic textbooks, like that of Strümpell.

Jaeschke (*loc. cit.*) who, in 1882, reported two cases of arsenical paralysis, argued that the symptoms were due to neuritis, because—

1st. Of the localization of the sensory or motor disturbance in the limits of certain nerves. In one of his cases there was a well-marked anæsthesia, limited to the regions of the median nerve.

2nd. The marked sensory disturbances, especially the peculiar burning pains, ataxia, and tenderness along the nerves.

3rd. Absence of the characteristic symptoms in profound spinal paraplegia, viz. of vesical weakness.

4th. Absence of the rapid and high grade atrophy which follows anterior poliomyelitis.

5th. The comparatively rapid and complete cure, despite a high grade of paralysis.

6th. The presence of herpes zoster.

7th. The electrical reactions, which are those of a mild form of peripheral paralysis.

8th. The marked tenderness of the paralysed muscles.

A. To these arguments I would add that of the presence of optic neuritis, as in Da Costa's case and one of my own.

B. The occasional arsenical monoplegias, much as that described by MacKenzie of one of the vocal cords.

C. The fact that arsenic may produce a perfect type of pseudo-tabes, like that from alcohol or diphtheria, and the fact that in these latter cases the trouble has been shown to be a neuritis.

I beg to enlarge upon this latter point.

It is very well known that there are post-diphtheritic paralyses of various types, and that among these is a form of ataxia or pseudo-tabes due to the diphtheritic infection. It was noticed first by Orillard, in 1836, and differentiated by Eisemann, and Jaccoud later. Many illustrations of it have been reported, and Buzzard, in his work, acknowledges that it may even be mistaken for true tabes. The only post-mortem made in a case of this kind was that of E. Mendel.[1] He found a well-marked neuritis, with slight central changes, indicating, perhaps, a beginning myelitis. The post-mortems made in other and ordinary forms of diphtheritic paralysis have shown, with very few exceptions, well-marked neuritis, with slighter central changes. Déjérine's cases are the most important in contradiction, and he found degenerative neuritis as well as myelitis.[2] On the other hand, Charcot, Vulpian, Lorain, and Lépine, Liouville, Leyden, P. Marie, Buhl, P. Meyer, and E. Gaucher, have all found decided evidence of neuritis.[3] So that it may be said with much confidence that the diphtheritic pseudo-ataxia is of peripheral origin.

Again, there is known to be an alcoholic paralysis proper and an alcoholic pseudo-tabes. The latter disorder closely resembles the pseudo-tabes I have described. It is very well settled that both these forms of alcoholic paralysis are due to a neuritis. The evidence is fully gone over in the articles by Dreschfeld[4] and by Schultz.[5] It is very probable, also, that in the rare cases in which lead causes pseudo-tabes the disturbance is a peripheral one.

That a pseudo-tabes may be caused by neuritis is shown again in a case reported by Déjérine.[6]

Several have described arsenical paralysis as resembling the subacute spinal paralysis of Duchenne. But Leyden cites two cases with post-mortem, one of Eisenlohr, and one of his own, to show that this is a multiple neuritis.

There are, then, two forms—often not very sharply defined—of arsenical paralysis, just as there are of the post-diphtheritic

[1] 'Centralbl. f. Neurol.,' 1885, No. 6.
[2] 'Arch. de Phys.,' p. 107.
[3] *Vide* E. Mendel's article, *loc. cit.*
[4] 'Brain,' Jan. 1886.
[5] 'Neurol. Centralbl.,' No. 21, 1885.
[6] 'Brain,' 1884, p. 558.

and alcoholic paralyses, viz. (1) arsenical paralysis proper, (2) arsenical pseudo-tabes. Clinically these various toxic paralyses have a close resemblance; indeed, arsenical pseudo-tabes is as nearly identical with alcoholic pseudo-tabes as well can be. Now since the latter is known to be due to a peripheral lesion, it is a legitimate inference that the former is so also. Until post-mortems are obtained we cannot get any nearer certainty.

We have at least shown, however, how entirely improbable is the dictum that arsenical paralyses are always due to a diffuse myelitis, although in some cases such lesion may exist. And we have shown another cause for peripheral tabes. The establishment of the fact, that arsenic as well as alcohol, diphtheria, lead, and probably other poisons and infections, may cause a disease closely resembling posterior spinal sclerosis, will do much to clear up the doubts that have of late attached to the unity and autonomy of that disease. Cases of locomotor ataxia reported cured, and cases of supposed posterior spinal congestion will have to be reviewed in the light of these facts. When Déjérine described as locomotor ataxia a case which now appears to have been one of alcoholic peripheral neurotabes; when Buzzard has diagnosticated as true spinal tabes a case of post-diphtheritic ataxia; and when Seeligmüller mistakes for a time a case of wall-paper poisoning for one of true spinal tabes, we may easily suppose that errors have been made by many others, especially those who have been so fortunate as to see patients with tabes cured.

Conclusions.

I. That a disease resembling locomotor ataxia may be caused by arsenic, given medicinally, absorbed from wall-paper or taken in a single dose.

II. That arsenical paralyses of this type, and arsenical paralyses of other types, are not due, as a rule, at least, to a diffuse myelitis, as has been taught, but to a multiple neuritis.

III. That arsenical paralyses, like those from diphtheria, alcohol, lead, and probably other infections and poisons, are of two types.

(*a*.) The ordinary mixed motor and sensory paralysis; the motor troubles and atrophy being more marked.

(*b.*) The pseudo-tabetic form in which there is no pronounced motor paralysis, but marked sensory troubles, and especially ataxia.

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As arsenical paralysis is not yet systematically treated of in text-books, and as no complete bibliography has been compiled, I have undertaken to collect the references. The task is made comparatively easy through the learned contributions of Imbert Gourbeyre, who, in a series of articles published in the 'Gazette Médicale de Paris,' 1858, and in an elaborate monograph, entitled "Des Suites de l'empoisonnement par arsenic," Paris, 1881, covered the ground quite thoroughly up to five years ago.

Arsenical paralysis was first referred to in the 14th century by P. Albano: "De Venenis eorumque Remediis." The fact that arsenic in toxic doses may cause paralysis was evidently known to most medical and toxicological writers who succeeded him, and was referred to by Forestus, 1550; Kircher, 1578; Weikard, 1626; Schroeder, 1641; Ramlovius, 1650; Tirnoeus, 1662; Wessfer, 1629.

In the 18th century we have the following references:

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Barrier: 'Journ. de Méd.,' 1783.

Pyl: 'Aufsätze u. Beob.,' viii., Berlin, 1784.

Hahnemann: 'Ueber Arsenik-Vergift.,' Leipzig, 1786.

Geoffroi, in 1741, first wrote systematically of the affection.

Cranz, in 1755, and Hahnemann also described the symptoms quite thoroughly.

In the 19th century the same class of reports was made, there being no careful attempts at analysing the symptoms, or searching out the true pathology, until within the past twenty years. The chemical and forensic sides of the subject have almost always been in the foreground, and no post-mortems are reported. I have used the abbreviations Chr. p. q. o. and l. o. for chronic paralysis, quick or late onset.

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Among 120 cases collected by Gourbeyre, the paralysis in over one-half was cheiro-podal, stopping at elbow and knee. In ⅓ there was paraplegia. In 4 cases hemiplegia. In 2 cases hemianæsthesia. In 3 cases of one limb. In one case of a vocal cord. Sensory troubles are always present, and precede the motor. Anæsthesia may follow in distribution of a nerve. Burning, and lancinating pains, and great muscular tenderness are characteristic. Desquamation of the epidermis often occurs. Herpes zoster has been noticed. Atrophy is often general, but in chronic cases may affect special groups of muscles. Almost all cases recover.

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The original view put forth by Scolosuboff, that there is a great proportional excess of arsenic in the brain and cord, has been abundantly refuted by many other observers (see above). It is now well determined that, in almost all cases, arsenic is absorbed by the different organs in about the order named: the liver, spleen, intestines and kidneys take up the most; the brain generally has only a trace, sometimes none at all, while the muscles take up a considerable amount.

COMA.[1]

BY CHARLES MERCIER, M.B.

In one of the latter numbers of the 'Medical Times,' Mr. Savory had an important and suggestive note calling attention to the vagueness of meaning which attaches to the term *coma*, and to the pathological and clinical diversity of the conditions to which the term is applied; and he sought to restrict its application to a particular class of cases. He would have the term *coma* associated with those cases only in which there is a state of insensibility from which the patient cannot be completely aroused, together with a tendency to death chiefly by asphyxia.

To me, this limitation proposed by Mr. Savory appears strictly scientific and thoroughly practical: scientific because these two conditions—insensibility and tendency to asphyxia—are invariably associated with other morbid conditions in a well-defined group, so marking a state as definite pathologically as it is clinically; and practical because the state thus characterized may invariably be recognised by a test that it is extremely simple and absolutely pathognomonic, being never absent in coma, and never present except in coma as thus defined.

Looking at the matter from a point of view different from that of the surgeon, I would substitute for Mr. Savory's expression, "insensibility from which the patient cannot be completely aroused," the expression "evidence of defect of consciousness." The meaning of the latter expression will doubtless be much less obvious than that of the former, but it is for the present purpose more convenient. When we say

[1] The descriptive portion of this article appeared in substantially the same form in the 'Medical Times' for March 28th, 1885. The pathological discussion is new. Both portions are inspired by the teachings of Dr. Hughlings-Jackson, who must be credited with whatever of value the article contains.

that a person is insensible, we make a statement which is scientifically unjustifiable. All that we are warranted in stating is that he gives no evidence of consciousness. To insist on accuracy in this matter is not mere pedantry, for those who are familiar with cases of insanity know that many people have appeared to be, and have been recorded as, "insensible," "unconscious," "not knowing what is going on around them," and so forth, when, as a matter of fact, they are fully alive to passing events, and were able subsequently to describe them.

The reason that the phrase "defect of consciousness" is substituted for "insensibility," is that the latter implies absolute unconsciousness; it is used convertibly with "loss of consciousness;" it implies temporary mental annihilation. But in very many cases of coma, while there is evidence that much consciousness is lost, there is also evidence that some consciousness remains. Such terms as "insensibility" and "loss of consciousness" imply and suggest that there is an abrupt separation between full mental efficiency and total mental nothingness, and that a person must be in either the one state or the other. But this is not the fact, and the phrase "defect of consciousness" seems better adapted to convey the notion, that between the full daylight of vivid mental activity and the utter darkness of unconsciousness there is a long twilight, in which consciousness is not wholly lost, but is defective in various degrees. Just as on the physiological side there is every gradation from full mental alertness through fatigue, lassitude, drowsiness, semi-somnolence, sleep with vivid dreams, sleep with faint dreams, to profound slumber; so on the pathological side there is every gradation from slight confusion, through dulness and many grades of coma, to deep "insensibility."

To Mr. Savory's doctrine that, in that state to which the term coma should be limited, this evidence of defect of consciousness is always associated with a tendency to death by asphyxia, I entirely agree; but again, looking at the matter from another point of view, this tendency to death by asphyxia —this failure of the respiratory process—appears but a part of the bodily condition with which the defect in consciousness is

associated. In deepest coma, when there is "insensibility" so profound that no amount of stimulation evokes any answering reaction, there is laboured respiration, and there is much besides. The respiratory defect is peculiar. There is a deep inspiration, followed by a long noisy expiration, and then a long pause. In other words, the respiratory centre does not act with its normal freedom. The small accumulation of carbonic acid, which is normally enough to excite its action, is no longer enough. A stronger stimulus is required. The effete ingredients must accumulate for four or five times their usual period before they reach the degree of concentration which can act as a stimulus to the sluggish centre. When the respiratory act does occur it is imperfect. Only the fundamentally necessary parts of it, the raising of the ribs, is well performed. All the accessory portions are imperfect, and the less essential they are, the more imperfectly are they performed. The diaphragm does not descend to an extent at all proportionate to the elevation of the ribs. The loud gasp that occurs even when the mouth is open and the tongue pulled forward, shows that the glottis does not dilate properly. Flaccidity of the soft palate allows the loud snore; imperfect action of the lips permits the purring sound. It has been said that the respiratory defect is but a part of the bodily condition. The skin is relaxed; it is flaccid and covered with moisture. If we pass a catheter the urine flows sluggishly; the bladder does not contract vigorously. Throw a bright light upon the eyes: the pupils will contract little or not at all. The pulse is very full; in other words, the arteries are flaccid—their muscular coat is relaxed. But there is much more than this. *The limbs are all paralysed.* It is generally supposed that the patient lies still because he is unconscious; but, as Dr. Hughlings-Jackson, in his Croonian lectures, has shown, this is nonsense. It is the commonest thing in the world for an unconscious person to move his limbs. The fœtus in utero moves its limbs before consciousness has come into existence. But no one, conscious or unconscious, can move a paralysed limb. The comatose patient lies still, it is true; but examination shows that the limbs are not the limbs of a person who lies still because he does not want to move. The muscles are

flaccid. If we raise a limb and drop it, it falls like the limb of a corpse. It flops down in a flail-like manner that does not occur in, and cannot be imitated by, a limb that is not paralysed.

Similarly, the head rolls from side to side under the influence of gravity, without check from any tone in the muscles of the neck. To sum up the bodily condition of the comatose patient: there is paralysis, more or less complete, of all the voluntary muscular system; there is incomplete paralysis of the muscular system of the skin, of the arteries, of the bladder, of the pupil; there is incomplete paralysis of the respiratory system; and the muscles of the heart appear to be the only ones that are not weakened in some degree.

If we examine patients whose coma is less profound, we obtain evidence of some remnant of consciousness. On violent and repeated irritation, the patient feebly moves a limb; he groans; under a strong light his pupils sluggishly contract; his face is less dusky; his pulse is less full; his diaphragm acts better. But he still snores; he is still incapable of strong movement; he cannot hold up his head. In this case there is some evidence of dim consciousness, and there is paralysis of less degree than in the previous case.

Patients who exhibit a less degree of coma are more easily aroused. Manifestations of consciousness occur under smaller stimuli, and are of more vigorous character. Pinch the skin and they swear; repeat the pinch and they strike out; throw a light into the eyes and the lids close. But their movements are feeble and are not sustained; they cannot sit up; they do not answer the simplest question; they cannot lift hand or knee against a feeble pressure. There is still evidence of defect of consciousness, and there is still paralysis.

There is a stage of mental confusion and of bodily weakness which is not coma—is not severe enough to be called coma—but is a still slighter degree of the same malady. In such cases the patient can talk, but he talks stupidly. He can answer simple questions, but is easily confused. He can stand, but he sways about. He can walk, but he reels. Here, again, there is evidence of defect of consciousness, and there is muscular weakness—there is some degree of paralysis.

The four stages described are connected by innumerable intermediate gradations, rendering the whole series continuous; but to whatever degree the coma has extended, the amount of paralysis present is always proportionate to the degree to which consciousness appears defective. The bodily defect and the mental defect proceed *pari passu*, and signify, it cannot be doubted, the same lesion. The various stages of coma may all be necessarily exhibited in the same individual, and are so exhibited to perfection in the coma due to alcohol.

It is to be particularly noted, and this observation is due to Dr. Hughlings-Jackson, that the paralysis is first a loss of *movements*, and only at last a loss of *motion*. That is to say, the finest, most delicate, and most elaborate movements, and those that are most associated with Will, are the first to go; while the simplest, broadest, most general movements, and those least associated with Will, are the last to be retained. It is under the operation of this law that the drunkard first becomes unable to write; then his articulation gets thick; then he spills his liquor; then he is unable to walk, and at length sinks under the table. But this is not all; *it is under the operation of precisely the same law that his breathing begins to fail, while his heart remains unaffected*, and that, as has been pointed out, the accessory parts of the breathing apparatus fail before the fundamental parts. The full pulse and the stertor are all of a piece with the inability to write and the thick articulation. They are due to an extension of the same process in the same direction in the same way; and this is why it is said that those cases of insensibility in which the tendency to death is primarily by asphyxia form a natural pathological group. To say that the tendency to death is chiefly by asphyxia is to say that the breathing is failing before the heart; and this is the natural termination of the long process of degradation which begins with the highest and most elaborate movements and spreads disastrously and continuously down to the lowest, simplest, and most fundamental. Follow the process to the bottom, and you find that the last point of attack must be the heart, and the penultimate point the breathing.

I have said that there is a test for coma which is absolutely

pathognomonic. This statement must be understood to exclude the pre-comatose stages of coma, and to apply to those stages only in which there is what is ordinarily termed "insensibility"—in which the patient is incapable of standing, or sitting, or answering questions; although it occurs in very light coma, and sometimes before the conjunctivæ become insensitive. The test to which I refer is one to which attention was drawn—as a symptom—both by Dr. Warner and myself, in the 'British Medical Journal,' about eight years ago. It is the movement of the two eyes independently of one another. About the time that the conjunctiva becomes insensitive, the eyes are noticed to have a peculiar unintelligent "far away" expression. If examined, it is found that this expression is due to the absence of any convergence to their axes. They are quite parallel. The next thing is, that they begin to move independently. Usually one rolls out while the other remains still. Granting the absence of previous strabismus, actual divergence of the eyes is absolutely certain evidence of coma. There are people who can move one eye through a small definite arc while the other remains fixed, but no one can voluntarily cause his eyes to diverge; and in coma the eyes, if watched, will *always* be seen to diverge. They do not always begin by diverging; they may converge; they may both move in the same direction—upwards or downwards, to right or to left—but they will not both move at the same rate and to the same extent. The deeper the coma becomes, the more independent become the movements of the eyes. The difference in their direction is often astonishing. One may be directed out and up, and the other out and down. One may be directed downwards, while the other is hidden beneath the upper lid. If the patient can be momentarily roused by severe stimulation, the eyes momentarily resume their parallelism; as he subsides into coma, so they fall back into independence. It matters not what the cause of the coma may be—whether alcohol, ether, chloroform, uræmia, hæmorrhage, a previous epileptic fit, a blow on the head, meningitis, cerebral tumour, or what not—if there is coma the eyes move independently; if the eyes move independently there is coma. I am unable to say whether the same thing occurs in normal

sleep, but it very likely does occur, sleep being physiological coma. From a diagnostic point of view, the importance of this symptom is evident. If only because it instantly negatives the possibility of hysteria, or malingering, it should always be looked for in light coma. So easy is it to recognise, that I have long been in the habit of teaching nurses and attendants to look for it, and to regard it as the first sign of danger.

Such being the clinical phenomena, the question for us is, What is the pathological process that these phenomena indicate? What are the changes inside the body to which these outward and visible signs are due?

Two characteristics of the phenomena are specially to be noted. First, that there are two sets of changes; and second, that these two sets of changes proceed concurrently. There are the bodily changes, and there are the mental changes. Difficult as it usually is to establish a parallelism between bodily and mental phenomena, here the parallelism is obvious. Both sets of changes are in the same direction. Both are in the direction of *enfeeblement*. Consciousness—so far as we can judge of it by its outward manifestations, and by the descriptions of those who have gone through the process—marches steadily from the light, through a penumbra of greater or less breadth, into the darkness of oblivion. As with the mental strength, so with the bodily strength. There is a continuous loss of muscular power, ending in complete powerlessness. The slight unsteadiness becomes a reel, the reel ends in a fall, the struggles subside, and the patient lies like a log, the picture of utter prostration.

There is a still further parallelism, and a very remarkable one. The enfeeblement, both in the one case and in the other, is not a mere enfeeblement. It proceeds not at random, but in an orderly march, obliterating one faculty after another, one function after another, in regular sequence. Ask a man who is just leaving a convivial dinner to settle with you the lease of his house, or a deed of partnership. He will naturally and certainly refuse. If you press him, he will say it is not a proper time; and if pressed further, will explain that to take him now is unfair, for to such an important and delicate

matter one must come with a clear head. The admission is, that the mind now is not as vigorous as it will be to-morrow morning. It is slightly enfeebled. In other words, by the fatigue of the day—by taking a heavy dinner, and so drafting off a portion of the blood-supply from his brain to his stomach, by imbibing alcohol, and so benumbing to a certain extent his highest nervous centres—*he has travelled the first stage on the road to coma.* Now note carefully the character of the enfeeblement. Our *convive* is still able to enjoy his chat and his joke, to pay his legal cab fare, and to scold his wife; but he will not trust himself to settle the precise terms of his will. What he is unable to perform are *operations of a high degree of complexity, delicacy, and precision.* Similarly on the bodily side. He can walk perfectly straight, he can light a cigar without bungling, and button his overcoat with ease; but when he tries to play billiards he finds his "hand is out." So that of bodily movements, as well as of mental operations, he has lost the most precise, the most delicate, the most complex.

As the coma deepens, the parallelism is maintained at successively lower stages. When the drunkard has reached that stage at which his articulation is thick and his words run into one another, he has on his mental side reached a stage of confusion of mind such that he no longer appreciates that the stake he is playing for is beyond his means. When the invading feebleness reaches his locomotor movements, and, overcome with languor, he staggers to a couch, his mental insufficiency has so increased that he mistakes the room for his bed-room and drops his boots into a cupboard in the belief that he is putting them outside his door. By the time that his bodily weakness has reached the stage of prostration, so that he is unable to rise, and by his utmost effort can only kick or strike out aimlessly and feebly, his mental capacity has reached a corresponding stage of diminution, he has lost all knowledge of his surroundings, and is merely confusedly conscious of the discomfort of being disturbed. When the prostration is complete, consciousness is lost.

Having regard to the strict and constant parallelism between the two sets of changes, the inference is unavoidable that they

are due to the same lesion. It is infinitely improbable that two separate lesions should begin at the same moment, follow precisely the same course, step by step keep time together, and culminate simultaneously.

Whatever the intimate nature of the lesion, the nature of the phenomena—their constant character of enfeeblement—indicates that the lesion must be of an enervating character: it must have on the functions of the nervous centres a weakening, arresting, obliterating, nullifying influence.

Seeing that at the very outset the most elaborate mental processes are affected, the starting-point of the lesion must be in that nervous region whose activity underlies these processes. Seeing that at the outset the most elaborate bodily movements are affected, the starting-point must be in that nervous region whose activity actuates these movements. Hence it appears that the highest of all nervous regions, viz., that which underlies the most elaborate mental processes, represents at the same time the most elaborate bodily movements. So that when the highest of all nervous processes are arrested, by this single pathological event is produced the double set of symptoms—the enfeeblement of consciousness and the enfeeblement of conduct.

At the next stage of the malady, the further enfeeblement of consciousness and the concomitant and parallel enfeeblement of movement indicate an extension of the original lesion. They indicate that, in addition to the loss of the highest nervous processes, there is now a loss of other and subordinate processes of a less elaborate and more fundamental character. As the lesion extends, all those nervous regions are at length put out of action which subserve, however faintly, the manifestations of consciousness; and consciousness is altogether extinguished. By the same lesion all movements classed as intelligent are obliterated. Should the erasing process descend still further in the nervous centres, it lays hold at length upon the last strongholds of vital activity. It stills the breathing; it stops the heart. Thus the whole double train of symptoms displayed in coma are accountable for by the gradual extension of a single lesion.

But the train of symptoms in coma is not merely double.

It is multiple. And each train of symptoms runs a similar parallel course, and exhibits the same series of gradations as those we have already considered. Herein lies an implication of profound significance.

At the very outset of the malady, at the very first step of the long journey which terminates in coma at the door of death, not only is there some enfeeblement, however slight, of mind, not only is there some enfeeblement of body, but there are other phenomena also. The face is flushed, showing diminished vascular tension; the eye is bright, showing greater fulness and increased secretion; the pulse is more rapid, showing altered cardiac innervation; the skin is moister; the renal secretion is augmented; the stroke at billiards, which now fails, requires not only a precise adjustment of ocular muscles and a precise movement of hand and arm, but also a firm attitude of legs, and a steady tension of the muscles of back and belly. At the very outset of the coma change there is an affection not only of the mind, not only of the most elaborate movements, but also *of every part of the body*. The entire organism is affected. And this affection is due to a lesion of the highest of all nervous processes. Thus we arrive at this most important conclusion: that the highest nervous processes, which form the substrata of the most elaborate mental operations, represent at the same time not only the most elaborate forms of conduct and muscular movements, but also *every part of the organism* in some degree.

It may be objected, that in the case supposed—that of incipient coma due to alcohol poisoning—the similar effects on various functions may be produced separately by the direct action of the alcohol upon various separate centres,—the vaso-motor centre, the cardiac-inhibitory centre, the renal secretory centre, the cutaneous secretory centre, and, for aught I know, the oculo-glistening centre and the billiard-playing centre. But this explanation will not hold water, for it will not account for the strict parallelism of the involvement of so many separate functions, nor will it account for the progressive and parallel involvement in coma from such a cause as cerebral tumour.

Moreover, we can reach the same conclusion by two other

very different routes; and the fact that we can do so, goes far to place the conclusion beyond question.

The first route is this. The highest nervous centres are the anatomical substrata of consciousness; and consciousness *in toto* is made up of object-consciousness and subject-consciousness—of that which is thought and that which thinks. Of object-consciousness, the physiological substratum or condition is the reverberation of the nervous currents debouching into the highest centres from the organs of sense, and so from the external world. Of subject-consciousness, on the other hand, the physiological substratum is, as Ribot has pointed out, the wash on the shore of the highest nervous centres of nerve-waves arising from the interior of the body. And if from the body, then necessarily from the whole body, or the subject-consciousness would be partial—erroneous, as indeed it becomes when, by the severance of a nerve-trunk, the currents flowing from any part of the body are arrested.

The second route is more direct. Let it be granted, that the function of the highest nervous centres is to adjust the organism as a whole to its environment; then must the whole organism be represented in these centres, or the adjustment will be erroneous—will be not that of the whole organism, but of a part only.

The establishment of this principle—that the highest nervous centres represent in more or less degree every part of the organism—is momentous, and leads to results of the very highest importance.

To the physiologist it is of value, since it demonstrates that the various functions of the encephalon are not segregated in separate encapsuled portions of the grey matter, and shows the inadequacy of that view which regards one localised portion of the brain as representing movements of the arm only, another of the leg only, and so forth. The doctrine of nerve-centres is rapidly becoming, like so many doctrines before it, a fetishism. Some years ago every obscure symptom was accused of being "reflex," and reflex action was regarded as the universal solution of every clinical problem. Reflex action was displaced by incoördination—a term which by its superior vagueness and sonorousness was still better qualified for the

post of Universal Cause. But incoördination has been deposed in its turn and beaten clean out of the field by the doctrine of nerve-centres, which is the most efficient fetish we have had yet. Is the gait affected? It is due to disease of the locomotor centres. Is there atrophy? The trophic centres are paralysed. Hypertrophy? The trophic centres are rampant. Glycosuria? The glycogenic centre is out of order. Ataxy? The coördinating centres (!!) have gone wrong. Have we a new and inexplicable symptom? Quick! invent a centre—two—three—half-a-dozen centres; destroy one, discharge another, and all is clear. For every motor disorder a separate centre is at fault; and when we study aphasia, we must first go back to Euclid and refresh our remembrance of geometry before we can hope to unravel the complicated diagrams in which the relative positions of the centres are demonstrated with such mathematical certainty.

Far be it from me to disparage the study of the localisation of functions in the nervous system. The discovery of this localisation is without doubt the most important event of this generation, and in the history of physiology ranks second only to that of the circulation of the blood. But while we admit, while we proclaim the localisation of function, let us remember to say that the localisation of representation is not absolute, but partial only. In the lowest centres of all the localisation is no doubt extreme. Such centres represent a limited part of the body very strongly; they represent little else, and that little but feebly. But in the highest regions each centre represents a large part of the organism preponderatingly, a still larger part in less degree, and the whole of the organism in some degree. And in the intermediate centres the representation is intermediate in character, a larger or smaller area being preponderatingly represented, and the halo of partial representation being larger or smaller, while the intensity of representation is less or more, according as the centre is more or less elevated in the hierarchy of the nervous system.

To the physician, and especially to the alienist, the establishment of the principle of the universal representativeness of the highest nervous centres is of profound importance. It clears up some of the obscurest and most recondite problems,

both of medicine and of insanity. There is an old and familiar observation, that insanity has many physical accompaniments. The skin is altered; it sweats unduly, or is unduly dry, or desquamates more freely than normal. The hair is altered; it is harsh and staring. The nails get ridges on them, or are even discarded. The digestive function is altered; the whole process of nutrition is disturbed throughout the body. The lunatic is a lunatic, as Bucknill says, to the tips of his fingers, and, as Darwin says, to the end of every hair. And we now see why. We know that when a muscle is paralysed it quickly atrophies—when the skin is insensitive it readily sloughs; in other words, derangement of the function of a nerve-centre modifies not only the function of the part represented in the centre, but the nutrition of that part also. And hence derangement of the highest nervous centres disorders not only that adjustment of the organism to its environment, which it is the special function of these centres to effect, but disorders also the nutrition of every part represented in them —that is, the nutrition of the entire organism.

In this doctrine we find, moreover, the explanation of those almost miraculous bodily changes which occasionally follow upon tremendous storms of emotion. Such a change, for instance, is the rapid blanching of the hair, completed it may be in a single night, nay, in one recorded case within an hour (*vide* "Data of Alienism," 'Jour. Mental Science,' Oct. 1884.) Such changes are the pigmentation of the skin and other phenomena alluded to in the article above mentioned. Of course the principle here upheld does not show us the actual mechanism or chemistry of the changes in question, but that is a detail. Granted that a modification of nerve-currents modifies the nutrition of the parts into which they flow (and of this there is no longer any question), and the general alteration of nutrition throughout the body consequent on a commotion in the highest nervous centres becomes, on this principle, no longer a cause for wonder, but a necessity of natural causation. In the case of insanity, the alteration of general nutrition is mild and long enduring, in correspondence with the slight and chronic derangement of the highest nervous centres which is its cause. In the cases adduced in

the article above-mentioned, the change of nutrition is sudden and astonishing in correspondence with the sudden and violent perturbation of nerve function on which it depends. Both cases are additional evidence as well as striking illustrations of the principle here advanced.

But what, it may be said, is the relevance of this discussion in an article on coma? How does it help to elucidate the nature of coma to show that the highest nervous centres in which coma begins, represent the entire organism? Why, in this way: that assimilates the subject of this article to other and widely different maladies, and shows in fact that *coma is a form of insanity.*

Let me not be understood to mean that every patient with incipient coma should be forthwith locked up in a lunatic asylum. The botanist who declares that an onion is a kind of lily, is not necessarily understood to mean that his bed of onions should be immediately transplanted into his flower garden. I speak not of insanity in the legal sense. But I wish to emphasise the doctrine that insanity is not a thing *sui generis*, and separated by an impassable gulf from all other maladies. If we want to unravel a tangled skein, we do not begin in the middle; we first find an end. And if we wish to comprehend insanity, we must not begin with the most striking and most histrionic cases, which first arrest attention, but patiently work backwards to the points, and they are many, at which the unique and inexplicable phenomena of insanity show their community of origin and of nature with those of other maladies, of which we have a better comprehension; and at these points we must begin our investigations. One such point of junction we have in coma, a disease which, from the preponderance and gravity of its bodily symptoms is included in the domain of the physician, but which is nevertheless in essence and in fact a form of insanity.

Coma is not merely a form of insanity, in the sense in which the delirium of fever and of belladonna poisoning are forms of insanity. It is much more. It is *the* form of insanity. That is to say, it affords in brief an example of the course that every case of insanity must go through. It is a very rapid insanity, it is true. Just as an epileptic fit exhibits all the movements

of a fortnight or a month jammed up into a few seconds of time, so an ordinary case of coma displays in a few hours the events that in another vesania may be spread over many years. And whereas in the case of ordinary coma the course of events is commonly distinctly progressive or retrogressive, so that a few hours makes a decided difference in the aspect of the case; in that prolonged coma which we call insanity the progress of affairs may be arrested at almost any stage, and the case present little discernible alteration for years together. From one cloud a drop of water may fall into the sea in a few seconds. From another cloud at the same altitude, a drop may fall on the top of a mountain, may spend years in a snowdrift, centuries in a glacier, years again in a lake, and weeks in a river before reaching the level of the sea. But no one doubts that the cause of the descent was the same in the two cases. So it is with coma and insanity. Coma is a fulminant form of insanity; insanity is a lingering form of coma. Pathologically, coma is loss of function of the nervous centres, beginning in the highest centres of all; in those centres which are the substrata of consciousness, which effect the adjustment of the organism as a whole to its environment, which represent, first and most, the most precise and elaborate bodily movements, and which represent in some degree every part of the organism. From this commencement the loss of function spreads steadily downwards, to nervous regions successively less and less concerned with the manifestations of consciousness; to regions which effect adjustment of a more and more general, simple and fundamental character; to regions which represent more and more restricted portions of the organism. Or, translating this march of pathological events into its concomitant course of symptoms, there is first a dimness of consciousness, with a misconception of the relation of the organism and its surroundings, which we call being fuddled, with an inability to perform the most elaborate acts, and with an affection of some kind of the entire organism. From this commencement the progress is from dimness of consciousness to total loss of consciousness, from misconception of the organism to its surroundings to complete absence of such conception. From an inability to

perform the most elaborate acts to an inability to perform the simplest acts, and then to an inability to perform simple movements; and from loss of complex and special movements to loss of simpler and more general movements, until at last nothing remains but the movements of the simplest parts of the breathing apparatus, and movements of the heart. Finally the breathing, due to compound tetanic muscular contraction, ceases, and nought remains but the isolated simple contraction of that fundamental morsel of the organism—the cardiac muscle.

Similarly, insanity is from a pathological standpoint a loss of function of the nervous centres, beginning in the highest of all, and often remaining restricted to these highest regions for years and years together. As in commencing coma, there is dimness of consciousness; as in that there is misconception of the relation of the organisms to its surroundings, which we now call delusion or illusion, or generally unsoundness of mind; as in coma, there is inability to perform certain acts of the more elaborate kind; and as in coma, there is some affection of every part of the organism. At this stage the insanity may remain. From this stage it may recover, precisely as the incipient coma of alcohol may recover if no more alcohol be taken. But suppose it do not recover. After weeks or months, or years, or almost a lifetime, one thing invariably happens. The teaching of experience is, that every case of insanity, if it goes on long enough, and is not cut short by some intercurrent malady, will invariably and inevitably at length *subside into dementia*. And what is dementia? What are its symptoms? In dementia the consciousness is dull; the vacant countenance, the hanging lip, the lack-lustre eye, the useless attitude of the hands, are all evidence that consciousness is defective. It is so defective, that if pinched or pricked the patient displays little or no concern, and he will let his toes char before the fire without moving his feet. Not only is consciousness extremely dull, but there is great lack of perception of the relation of the organism to its surroundings. The dement who has sat in the same chair every morning for months, and who has been taken from that chair to a particular place at a table half-a-dozen yards off

three times a-day during the whole of that time, yet cannot, unaided, find his way from one customary seat to another. Every act of even a moderate degree of elaborateness is lost to him. He cannot even cut up his food. With difficulty and imperfectly can he carry it to his mouth. Not only this, but those movements that he can perform are uncertain and feeble. In other words, there is some paralysis. Together with this greatly defective consciousness, this profound lack of perception of the relation between himself and his surroundings, and this inability to perform acts of even moderate precision, there is a profound affection of the entire organism. The skin is sallow, earthy looking, sweaty or glazed; the hair is harsh; the bowels are constipated; the sphincters are not under control; the hands and feet are blue and swollen; the saliva is increased and altered. Compare these symptoms with those of a moderate degree of coma. They are marvellously similar. Allowing for differences due to difference in the rapidity of the malady, they are identical.

Finally, notice the termination of insanity. The majority of chronic cases are terminated by intercurrent maladies; but the normal and inevitable end of every case of insanity that does not recover, and is not cut short by a quasi-accident, is in coma—true, profound, unquestionable coma. Every case of acute delirious mania that ends fatally, ends in coma. Every case of dementia is liable at any moment to fall into coma, and many cases do so end. Even where they do not so terminate, it is common for dements to have little lapses into apparent unconsciousness, with still greater loss of power; and these attacks, which are manifestly of the nature of coma, are as manifestly exaggerations of the patient's usual condition of dementia.

Should it be objected that the over-action, the shouting, the restlessness, the furor, the eagerness of mania are fundamentally different and opposite in nature to the quietude, the prostration, the paralysis, of coma, I say that the comparison is wrongly made. The over-action of insanity must be compared, not with the stuporose stage of coma, with which it does not correspond, but with what I have called the precomatose

stages of coma, with which it does correspond. Taking once more for illustration the coma due to alcohol, the parallelism between its stages and the stages of insanity, between its forms and the forms of insanity, is most striking, most unmistakable, nay, almost grotesque. Before the maniac subsides into dementia, he may be violent and ferocious; and before the drunkard subsides into somnolence, he may be quarrelsome and pugnacious; or the one may be melancholic, and the other maudlin; or the one have grandiose delusions, and the other be a braggart; or the one become lascivious, and the other amatory. It may be laid down categorically that there is no form of insanity whatever that may not have its counterpart in a case of drunkenness. How often have not the faulty articulation, the jovial aspect, and imperfect gait of general paralysis recalled to mind the early stages of drunkenness? The resemblance is not accidental, it is a fundamental similarity of nature, which of necessity exhibits itself upon the surface also.

Whether in origin, or in course, or in termination, coma and insanity are pathologically and symptomatically identical, and the only important difference between them is the difference in duration.

If this be so, then I maintain that we have made a considerable step forward, and gained a very important position from which to study both coma on the one hand, and insanity upon the other. The great duration of so many cases of the latter malady, tends to obscure their stages, and to obliterate from our minds their general course. We may know a patient for years, and know him only as a maniac, a melancholic, or a dement; but if we investigate his whole history, and see it to the end, we shall see that whatever form his insanity may most prominently and for the longest time display, in essence and in fact it is a case of coma. One stage may be enormously prolonged, and another evanescent or apparently absent; but the pathological processes, however various in duration, and however different in proximate nature, run in all cases the same ultimate course; they begin by ablating the function of the highest of all nervous regions, and they spread downwards as a mist rolls down a mountain, obliterating in succession the

functions of the various nervous regions in their order from the highest to the lowest.

It will be remembered that the highest nervous processes are the latest and least organised, and that the lowest processes are the longest and most completely organised; so that the process which obliterates the nervous functions in their order from the highest to the lowest, is a reversal of the process of Evolution,—a disorganising of parts in the inverse order of their organisation. Hence, by taking the view of Coma that is here advocated, we are able not only to assimilate this malady to the superficially-different malady Insanity, but to bring both together as typical and regular examples of the operation of one fundamental law—the Law of Dissolution.

A CONTRIBUTION TO THE PATHOLOGY OF DREAMS AND OF HYSTERICAL PARALYSIS.

BY CH. FÉRÉ, M.D.,

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FALRET, Moreau de Tours, Brierre de Boismont, have spoken of hallucinations that begin during sleep and are reproduced for several nights consecutively, and end in being finally accepted as realities during day-time. Faure[1] and Richter (of Pankow)[2] have given cases in which dreams have been the starting-point of persistent diurnal deliriums. Suicidal or homicidal impulses have been observed after two or three nights of dreaming; occasionally a single dream is enough to set up a mental disturbance that manifests itself the next day.[3] Lasègue[4] has insisted upon the relations between dreaming and alcoholic delirium. I have had myself[5] opportunities of showing the analogy that exists between hysterical and alcoholic delirium in connection with the dreaming state.

Nor is it with delirium only that dreaming has intimate connections. Nothnagel and others have justly remarked that dreams may play an important part as a determining cause of epileptic attacks. The nocturnal fits occasionally observed in hysteria are as a rule caused by terrifying dreams, usually connected with the moral shock that determined the first seizure in the patient. In a case I saw at the Salpêtrière, the first hysterical attack was caused by a dream of alcoholic

[1] Faure, "Etude sur les Rêves morbides;" ('Arch. Gén. de Méd., May, 1876.')

[2] Richter, 'Arch. f. Psychiatrie,' xiii. 1.

[3] R. Ball, "Des Rêves prolongés" ('France Médicale,' 1886).

[4] Lasègue, "Le Délire alcoolique n'est pas un délire, mais un rêve" ('Arch. Gén. de Méd.' 1881).

[5] Féré, "La Medecine d'imagination" ('Progrès Medical,' 1886, No. 37).

origin. The young woman, who had never before shown any signs of hysteria, was made to drink to excess one day by her brothers as a joke. She fell into a deep sleep, during which she had frightening dreams of fantastic animals going to devour her; without awakening, she entered a hysterical state that lasted two days, and followed by frequently repeated attacks. I wish in this paper to call the reader's attention to a peculiar accident, resulting from a terrifying dream in a hysterical patient.

Hysterical Paralysis after a Dream.—Aphasia with Paragraphia. Blepharospasm, &c.

Eugénie P. came to the out-patients' room at the Salpétrière on the 11th of September, 1886. Her mother, aged 40, is corpulent and quick-tempered, though she denies having ever shown any nervous symptoms; a minute cross-examination reveals nothing further. The father is not related to his wife, but is of nervous temperament, irritable, but with no marked neurotic history. He is in the wine trade, and his sobriety is problematical. No full account of the collaterals can be obtained. There have been three children; the eldest, aged 18, is a girl in very good health. The next is our patient; the third was a boy, who died of meningitis at 18 months.

E. P. is 14 years of age. She came to term and spoke and walked at about 14 months; was clean early, and had no convulsions nor any other early nervous symptoms. She suffered from so-called "hemicrania" as early as between her sixth and tenth year; these attacks were violent enough to suggest meningitis, and she seems to have had delirium after several short feverish ailments. She also was subject to nightmare and nocturnal terrors. The catamenia appeared last January, and are said to have been regular till July. The period of August was missed without any concomitant symptom, though since that time E. P. has had more restless nights; she dreams more, and is rapidly growing taller. In the night of the 20th to the 21st of August, E. P. dreams that she is pursued in the Place de l'Odéon by men who want to stab her; she runs with all her might, and at last escapes them. She awakes exhausted and bathed with perspiration. The next day her mother noticed while walking out with her that the child's legs were giving way at intervals, though she did not complain of fatigue. The dream occurred again the following night; and the next day, Sunday, the phenomena observed before were still

present; in addition to these the child was inattentive, not answering questions as usual; at times during the day she appears frightened, and says that it is her dream which is coming on again.

The same dream renews itself during the following night; the patient is again awakened in the same way, perspiring and exhausted. The weakness of the legs becomes more marked, she falls several times in the day. Day dreaming becomes more marked; the terror movements are more frequent and intense. She sees men pursuing her, and hears them calling out to her; she feels them seizing her hair. At times she breaks or drops some object she is holding in her hand, in order to prevent their taking it away.

The parents make her take walks as a means of distraction, and improvement of the sleep. But they find that this going out becomes impossible on account of the patient, who does not complain of any fatigue in her legs, giving way and collapsing to the ground.

This condition of diurnal and nocturnal dreaming persisted without any change till the 7th of September, when it diminished in intensity. E. was no longer terrified during the day, was calm and enjoyed sleep in the night of the 9th. The weakness of the legs appears, however, to be on the increase. During the morning of the 10th her sister calls her to try on a dress on the floor above. She rushes upstairs, but on reaching the top her legs cease to obey, and she sinks helpless to the ground. From this moment the limbs remain flaccid till the evening, when she begins to make a few inco-ordinated movements. She moreover appeared to be unconscious of her condition. She had no dreams during the next night either.

On the morning of the 11th the patient is brought to the Salpêtrière, two persons carrying her under the arms. E. P. is rather tall for her age, with a dark, dull complexion, giving one at first the impression of a good constitution.

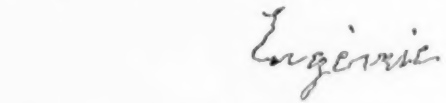
When at rest the physiognomy appears regular, but on closer inspection it is noticed that when she laughs the mouth deviates a little to the left; moreover when at rest, the naso-labial fold is more marked on that side. It seems as if the face be a little smaller on the same side. The two irides are of the same colour, but the pupil on the right is somewhat smaller. There is some ovarian tenderness on the right, as well as hemianæsthesia, slight in the arm, very marked in the leg. The special senses are likewise involved: she hears the watch at a less distance, taste is less developed, smell and sight less acute; the visual field, likewise, measured by a rough test, seems to be a little contracted on the

same side. She does not feel so well on the left leg as on the left arm. E. is incapable of standing, and collapses as soon as the support is withdrawn. The knee-jerks are exaggerated, especially on the right. When she sits, the toes hang downwards. The tendon-reactions at the right wrist are lively, whilst the dynamometer shows 18 on that side and 24 on the left.

When, being supported, she is made to perform the movement of walking, the foot is seen to advance; but when the heel is near touching the ground, a sudden spasm of the calf occurs that causes the toes to strike the floor, and the heel comes down an instant afterwards. This kind of "dicrotism" is noticed at every step. I prescribe a tonic regimen, three grammes of bromide at night, as well as rest and two daily sittings of passive movements of ten minutes each. These passive movements are carried out while the patient is reading aloud, so as to avoid the resistance of the gastrocnemii, which still are influenced by the will. On the 15th of September E. is seized with a fit of choking and crying, loss of consciousness, and a few spasms, followed with abundant micturition and defecation. On the 20th she comes again to the consultation. She has had no more dreams, and has recovered her usual temper. She walks on the arm of her mother, though the legs are still very weak, and the dicrotism still persists. The same treatment is continued, with cold douches to the trunk and limbs for half a minute every morning at the same time. On the 27th the improvement is still more marked. E. can walk by herself in the room, but the movements of the hips show that the trunk muscles are needed to supplement the yet paretic muscles of the legs; the patient has thus a dancing gait. Same treatment. Moreover, walking exercises to command, twice a day for fifteen minutes. On the 4th of October E. came on foot, without assistance, from the station to the hospital, a distance of about 300 metres. For some days past she goes out a little, walks by herself, though still with the same gait.

On the 9th, after her douche, her mother wanting to go out undresses her and puts her in bed, so as to prevent her from going about during her absence. E. enters into a fearful passion, shouts and talks wildly for two hours; after which she falls into a deep sleep. When her mother wakes her up on returning at midday, E. cannot say one word. She understands what she hears, moves her tongue properly, but no sound issues from her mouth. They try to make her write, but she cannot, though the movements of the fingers are perfect; she tries to trace the form of the letters, but is unable to make a single one regularly. In the course of the evening she begins to articulate a few sounds. On the 11th of

October, when she comes to the consultation, the patient talks in a jerky way and misses syllables; her tongue is the seat of choreiform movements. Though able to do fine needlework, she has the greatest difficulty in writing even her own name; she hesitates before beginning to write a letter and traces it tremulously. She walks much better, is able to run; the peculiar move-



ment of the hips is still noticeable, but the dicrotism is present only on the right side, which is weaker, and has more lively tendon-reactions. *October 18th.*—The catamenia have lasted three days, and were as abundant as in the past. Speech and writing have returned, the gait is much more normal. *Oct. 25th.*—Walking still further improved; the right leg only gives way a little after a long walk. Yesterday, however, after a fright caused by the fall of a piece of furniture, another symptom was developed, viz. a spasm of the eyelids, which move sometimes as often as 100 times in a minute. This spasm is accompanied with a convulsion of the superior rectus which remains contracted for fifteen or thirty seconds at a time; the eyeball is drawn completely upwards, so that vision is impossible. This spasm disappears within two days.

I saw the patient again on the 18th of November, when every trace of the symptom had disappeared except some right hemi-anæsthesia.

This case of paralysis after a dream deserves to fix our attention. It enables us, I think, to give another explanation of the origin of the so-called psychical paralyses as described by Profs. Russell Reynolds, and Charcot. It is assumed that these paralyses arise from an idea or suggestion; in other words, that the motor trouble comes on after its mental representation. This theory has even been applied to paralyses from traumatic causes. The circumstances under which the symptom has appeared in my patient, seem to me to support the theory of paralysis through exhaustion which I have propounded for traumatic paralyses.[1] The weakness of the limbs is really produced by the gradual influence of

[1] 'Bull. Société de Biologie,' 1886.

fatigue due to the exhaustion of the motor centres through a rapid succession of discharges ineffectual to produce movements. This same patient has moreover offered us another example of a fatigue paralysis, when she became incapable of phonating after the discharge of another centre.

I may add that, by calling forth dreams of running in hypnotisable subjects during their normal sleep, I have been able to produce similar paralyses that were accompanied by the same "dicrotic" gait, which we saw was due to the preponderating action of the flexion. In a certain number of cases of so-called psychical paralysis, the symptom supervenes after protracted cerebral work, and not after a subjective representation of fatigue or loss of power. Let us notice also in this case the disturbance in the writing which is an exceptional symptom in the history of hysterical asphasia. But the chief object of this paper is to draw attention to the influence of dreams in the development of certain psychical troubles. Our observations tend to show that dreaming, and especially repeated dreaming, must not be considered as an indifferent phenomenon, but often constitutes the opening scene of a morbid drama, and as such deserves the attention of the physician.

ON CHANGES IN NERVOUS SYSTEM AFTER AMPUTATION OF LIMBS, WITH BIBLIOGRAPHY AND RECENT CASE.

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THE changes which occur in the nervous system after the amputation of a limb, and which are probably due to the disuse of the member, are both interesting and instructive, as giving some slight clue to the functions of various nervous elements. Though an apparently trivial subject, still its literature is both full and fairly exhaustive, and this paper is intended rather to epitomise the results obtained up to the present, than to furnish many new facts. The changes noted in the peripheral nerves and cord, and published before 1880, were collected together by Dreschfeld in a paper entitled "On Changes in the Spinal Cord after Amputation of Limbs."[1] From this I have obtained the earlier references, looking up such of the cases as I had access to.

HISTORY.

Bérard, in 1829, found atrophy of the anterior roots supplying an arm which had been amputated. *Vulpian*, in 1868, reported two cases; one in which amputation was performed forty-seven years before death, where the cord was lessened in size both in grey and white matter on the amputated side, but without alteration of the cells; and a second (amputation twenty years ante mortem) with atrophy of the anterior white and grey matter, and of the motor ganglion cells, together

[1] 'Journ. Anat. and Physiol.' 1880, xiv. p. 424.

with granular degeneration in the anterior grey matter. In 1869 he again reported two cases with changes in the posterior white and grey matter; also a third case (amputation a year and a-half ante mortem) with changes in the anterior grey and white matter, but none posteriorly. Experimenting on animals, he found that tearing or irritating peripheral nerves, caused ascending degeneration in the cord. *Hayem* (1873) obtained the same results, but *Cruveilhier* and *Türck's* experiments were negative.

Dickinson reported three cases in 1869. The amputations were all of more than twenty years' standing. In one there was a neuroma at the cut end of the sciatic. This showed a granular appearance from the presence of minute circles (with no difference of grey or white matter), separated by structureless material giving a very pink section after staining; there was much connective tissue round, but both sciatics were the same to the naked eye. The changes found in the cord were, diminution of the grey matter of the posterior column (especially the anterior extremity), wasting of the nerve roots, especially the posterior; all being found on the side of the amputation. There was found no change in the cells. He points out that Waller's theory of degeneration does not hold in these cases. His diagrams only show relations of size. *Lockhart Clarke* examined some of the last-mentioned cases, and found atrophy of the postero-lateral cell group of the anterior horn. *Friedreich* found the cord normal after an amputation of twelve years' standing. *Erlenmeyer* (1872) found the nerve fibres on the amputated side smaller than in the opposite limb. *Dickson* (1873) noticed atrophy and diminution in number of the cells of the anterior horn, especially in the postero-lateral group in a fifteen-year-old amputation of the leg. *Genzmer* found atrophy of the large cells of the anterior horns in the lumbar region (amputation of right thigh thirty-three years ante mortem). His plate shows also marked atrophy of all parts of the right side of the cord. *Leyden* gives three cases, all with corresponding atrophy of the cord, especially in the external part of the grey matter. *Dreschfeld* (1880) reported a case (amputation of left thigh fifteen years ante mortem) showing an amputation neuroma. There was no

change in the sciatic, nor in the anterior or posterior roots; the brain was normal. The left side of the lumbar cord showed few cells, and these were atrophied but not pigmented; they had no branches, and many no nucleus. At the external lateral border of the anterior horn, there was granular material with fibrils surrounding the cells, and staining well. Higher up, the groups had the full number of cells, excepting the intermedio-lateral group, which was still atrophic.

Hayem (1876) found fine fibres in the nerve stump grouped into bundles. He concluded that, in the place of degenerated nerve-bundles, whole bundles of fine nerve-fibres are built up. He found the new fibres larger in a twenty-four-year-old amputation than in one five years old. *Déjérine* and *Mayor* (1878) found an atrophy of nerve fibres in the cut nerve. This atrophy decreased upwards towards the cord, and disappeared completely at a certain distance above the amputation level. In the cord they found atrophy of grey and white substance in all parts on one side, without change of the nerve fibres and without decrease of the ganglion cells generally. But in one case of thigh amputation there was in the mid-lumbar region a diminution of cells to the extent of one-third, especially in the inner anterior group of the anterior horn. They found no microscopic changes in the nerve roots. *Erlitzky* amputated the limbs of young dogs and found lessening of the posterior root, of the posterior columns and posterior horn, with diminution in number and size of some cells of the anterior horn of the same side. *Edinger* noticed atrophy of the whole half of the spinal cord in a case of intra-uterine amputation of the hand, but the ganglion cells were unaffected. Later, he found in two cases of amputation only a slight atrophy in the lumbar cord.

Hale White and *Edmunds* in 1882 showed amputation neuromata to the Pathological Society. They found that the first change in the coiled bundles of nerve fibres at the end of the nerve was an ingrowth of delicate connective tissue from the perineurium. This caused sclerosis of the bundles, which then underwent fatty degeneration. Some of the fibres remained healthy, these being probably connected with parts of the limb still remaining. In one case (amputation of

thigh) there were changes in the tractus intermedio-lateralis in the lumbar region only.

Hayem and *Gilbert* (1884) publish a case of amputation of the forearm just below the elbow forty-seven years ante mortem. The brain was normal. There were neuromata on the ulnar, median and radial nerves in the stump. These were composed of a large number of small nerve-fibres, formed of an axis-cylinder covered with a thin layer of myeline and grouped in fasciculi. They were much intertwined, but for the most part the direction was the same as the fibres of the main nerve-trunk till they approached the periphery of the neuroma, where they were looped back. The relation to the healthy fibres seemed to be, that at different levels the healthy fibres perished and were each replaced by a number of small thin nerve fibres, but the actual point of continuity between the large and small fibres was not observed. The neuromata appeared therefore to be due to a regenerative process, new nerve-fibres growing from the cut end of the nerve. The affected radial was of diminished size, but the median remained the normal size. In both, however, the nerve fibres were atrophied, and there was a growth of interstitial connective tissue. The ulnar was increased in thickness and here were found two forms of nerve fibres: (*a*) large isolated fibres with a fine axis-cylinder and thick coating of myeline, accompanied by a longitudinal band of connective tissue; (*b*) fasciculi of small fibres with a comparatively large axis-cylinder and thin coating of myeline; the interstitial connective tissue of the nerve was increased. The anterior and posterior roots of the brachial plexus were atrophied, and contained more small fibres than normal. In the cord the posterior columns of the affected side were smaller, but for the other columns the reverse was true. There was atrophy of the grey substance (particularly at the level of 7th and 8th cervical and 1st dorsal nerves), affecting chiefly the cells of the anterior horns.

Dudley gives a case of amputation of the left leg of a general paralytic twenty-one years ante mortem. I have to thank him for his great kindness in allowing me to see his specimens. The brain was fairly symmetrical, excepting that the right

ascending frontal convolution was somewhat smaller than the left. He is the only one to note the microscopic appearance of the cortex in these cases, but he found no change which could not be accounted for by the general paralysis. The cord was healthy in the cervical and dorsal regions. In the lumbar region the left anterior cornu was much reduced in size, and the cells were less numerous and smaller, especially in the antero-lateral and postero-lateral groups. Many cells were pigmented. The left posterior cornu was slightly reduced. The left anterior nerve-roots within the cord were reduced in number, and in some the axis-cylinder was broken up and granular. The left anterior crural nerve was the only one examined. It showed very fine fibres, scattered irregularly between healthy fibres in groups which stained deeply; the diminished size seemed principally due to the attenuation of the medullary sheath. There was much adipose tissue between the fasciculi.

Just as I was finishing my own investigations, Dr. A. de Watteville kindly forwarded me 'Fortschritte der Medicin,' No. 23, Dec. 1st, 1886. This contained an important paper by *C. Friedländer* and *F. Krause* on the changes in the nerves and spinal cord after amputations. They give a bibliography of seventeen references, five of which I had not hitherto known of, and which I have added to my bibliography. It is somewhat curious that my method resembled theirs in almost every detail; but I shall have occasion to criticise their conclusions. They investigated eight cases (of from 3 months to 10 or more years' standing). They found a degeneration of the cut nerves quite different from the Wallerian degeneration. The peripheral nerves showed normal bundles broken up by atrophied bundles; the latter were very small, and separated by much wider spaces. They contained atrophied fibres with faint outlines and completely lustreless, occasionally a normal glistening medullated fibre being seen amongst them. The atrophied fibres did not give a blue coloration with Weigert's copper hæmatoxylin stain, but only a yellow colour, showing the absence of myeline. With aniline-blue and ammonia-carmine, the smallest circles showed a central point which was probably the remains of an axis-cylinder, but

this was not noticed in longitudinal section. There was in the atrophied bundle a considerable increase of nuclei. No new fibres were recognized, the atrophied fibres appearing equal in number to the formerly existing medullated fibres. These changes were found 3 months after amputation, but the fibres were not so much lessened in size as in cases some years subsequent to amputation. As the cord was approached, the number of normal fibres increased, these of course coming from the parts still remaining. They examined both anterior and posterior roots (central to the ganglion on the latter), and found no change, except perhaps a slight diminution in the number of fibres of the posterior root. The spinal ganglion contained many atrophied fibres. The piece of sensory nerve, however, between the ganglion and the junction with the anterior root was almost entirely atrophied, only a few normal fibres remaining. In the cord they found a decrease of the posterior column, also slightly of the posterior horn, but no affection of the nerve fibres. There were no changes in the antero-median, antero-lateral or central groups, but marked decrease in number of the cells of the postero-lateral group and of the vesicular column of Clarke. The differences in the latter, however, were slight as compared with the former (5·3 : 7 in Clarke's col., 1 : 2·5 in the postero-lateral group). The total decrease in size of half the cord was entirely due to the diminution of the posterior column and horn and of the postero-lateral group. They conclude that the changes only affect the sensory sphere, that only those sensory fibres connected with touch-corpuscles and end-bulbs are atrophied (a case of amputation near the ankle showing as many atrophied fibres as one through the thigh), and finally that the cells of Clarke's column and of the postero-lateral group are sensory in function. They promise in another paper to give their explanation of the atrophy of the fibres. I have quoted this paper somewhat fully, as the conclusions are so much at variance with generally accepted opinions.

The remaining cases relate only to the changes which have been observed in the brain. *Féré* and *Mayor* (1877) describe the brain of a man whose leg had been amputated thirty years

before death. In 1878, *Ch. Féré* published a paper entitled "Note sur les Cerveaux d'amputés," and *A. Mossé* one entitled "Note sur un Cerveau d'amputé." Unfortunately I have not been able to see any of these papers, so cannot give particulars. *Oudin* (1878) gave a case of a woman who had an injury to the right leg sixty-six years before death; it at once became useless and remained atrophied, and she spent the rest of her life in the sitting position. After death the brain was found to be asymmetrical, the left first frontal convolution was narrowed posteriorly, nearly rectilinear and without sinuosities; the anterior part of the left superior parietal lobule was also atrophied, and the convolutions were small. The ascending parietal convolutions were narrow, but more so on the left side; the left fissure of Rolando was shorter and more anterior than the right. No other changes were noticed either in the brain or cord. He adds that such an atrophy of the brain would probably result only if the limb were rendered useless in infancy. *Charcot* (1878) showed a photograph (to a Society at Paris ?) of a brain thirty-eight years after exarticulation of the shoulder. There was no difference on the two sides In this case *Déjérine* and *Raymond* found in the cortical region of the cord on the right side, a sclerotic atrophy of the lateral bundle, not reaching the medulla (was this Gower's tract?). *Féré* had a few days before this communicated a similar case to the Société de Biologie. *Luys* mentions a case of amputation of thirty-five years' standing, causing atrophy of the brain. *Sanders* (cited by Charcot in "Les leçons sur les localisations") gives cases of brain atrophy associated with infantile paralysis. *Colard* and *Landouzy* give similar cases. In these, however, the question arises whether the brain atrophy was due to the disuse of a limb, or whether the cases were not rather spastic hemiplegia of infancy, the brain lesion being primary. *Pitres* in 1877 contributed an article to the 'Gazette médicale de Paris,' on partial atrophies of cerebral convolutions, consecutive to old amputations of limbs on the opposite side. *Varigny* (1879) has a note on a case of amputation as pointing to the localisation of cerebral function. *Bourdon* gives a case in which the left arm had been amputated forty years before death, this being followed by progressive paralysis of the left

leg. He found an extensive atrophy of the motor zone of the right hemisphere, with a diverticular enlargement of the lateral ventricle towards the atrophic spot. *Gowers* (1878) reports a case of congenital absence of the left hand. He found a marked difference in width of the right and left ascending parietal convolutions about the middle third, the diameter of section of the former being ·35 in., and of the latter ·65 in., no other brain abnormality being noticed. This I mention as being somewhat allied to amputation cases, though of course the relation of cause and effect is here not so clear.

Recent Case.

I have recently examined the changes in the nervous system of a patient who died in the West Riding Asylum, Wakefield.

George W——, admitted Jan. 16th, 1875. æt. 35, was a labourer. He had been temperate, but had been insane for twelve months before admission. His mental symptoms were somewhat peculiar, but he was almost certainly an anomalous case of general paralysis with long duration, dementia being the chief feature. The left thigh was amputated through the lower third on Nov. 14th, 1879, in consequence of caries of the bones round the knee-joint. The wound was slow in healing, but no mention is made of him having suffered any special pain in the stump. He gradually became more demented, and died from phthisis April 2nd, 1886, 6½ years after the amputation. At the autopsy there were found thickened cerebral meninges, and excess of cerebro-spinal fluid, but there were no adhesions. The cerebral convolutions were fairly complex, but atrophied; there was no marked difference on the two sides. Pons and medulla appeared healthy, but were unfortunately not reserved for further examination. The lungs showed tuberculous pneumonia.

Special Examination.—The spinal cord macroscopically showed no changes in the cervical, dorsal, or upper lumbar regions. In the middle and lower lumbar regions, however, the cut section of the left half was much smaller than that of

the right half. Both white and grey matter, but especially the latter, appeared to be affected; the left anterior nerve-roots were smaller than the right, but no difference was noticed in the posterior roots. Microscopically, the only changes seen in the cervical, dorsal, and upper lumbar regions were pigmentary degeneration of a few cells in the anterior cornua, and a slight increase of connective tissue in the lateral and posterior columns, both changes being easily accounted for by the general paralysis. No unilateral changes were seen. The cells of Clarke's column were carefully examined, and found very unequal in number on the two sides in any one section, but the decrease was about as often on the right as on the left side, so that it could not be due to the amputation. To settle this important point, I examined this region in other cords, and I found the same great difference in number in any one section, some sections showing more cells on the right, others more on the left side. In fact, as these cells probably are connected with viscera, there does not seem to be the same necessity for them to be equal in number in any plane, as if they supplied two similar limbs.

In the mid-lumbar region the left half was smaller in all parts, excepting the anterior column. The lateral column was slightly decreased, the posterior more so, and especially at its anterior end; consequently the posterior commissure was inclined forwards from left to right. Both the anterior and posterior cornua were smaller in all diameters, the nerve cells of the latter having fewer processes. The nerve cells of the left anterior cornu stained only faintly, were rounded and possessed few processes; they were mostly loaded with yellow pigment. With a high power faint outlines of much degenerated cells were seen. The neuroglia was more granular and more deeply stained. The cells of the right anterior cornu were normal, excepting that a few were slightly pigmented. The qualitative changes of the cells on the left side were accompanied by great quantitative changes. Six sections were taken indiscriminately, and the cells of each group counted, the following remarkable results being obtained:

No. of Section.	Left.						Right.					
	1	2	3	4	5	6	1	2	3	4	5	6
Antero-Intern. Group	13	11	8	14	7	6	22	19	7	10	8	5
Antero-Lateral „	6	7	7	6	11	8	9	17	8	13	15	6
Central „	6	6	8	6	4	8	7	17	19	9	6	13
Postero-Lateral „	8	7	4	4	4	4	21	12	24	23	10	22
	33	31	27	30	26	26	59	65	58	55	39	46
Total . . .	173						322					
Average . .	28·8						53·6					

All the above groups are seen to contain less cells on the left side, and this is especially the case with the postero-lateral group, and after this with the central group.

The filum terminale showed the left cornu (especially the posterior) to be smaller, but the posterior columns were much the same size.

The Nerves.—The sciatic and anterior crural nerves on each side were examined. Both sciatic nerves were of about the same consistency, but the area of section left was slightly larger than that of the right. This seemed to be due rather to an increase of the bundles themselves, which had a more fatty and less solid appearance on the left side. The same applies, in a much less marked degree, to the anterior crural nerves. At the end of the left sciatic was a small nodular mass (about $\frac{1}{2}$ in. × $\frac{3}{8}$ in.) covered with connective tissue; this was evidently an amputation neuroma. A much smaller one was found at the end of the anterior crural. The sciatic neuroma was cut longitudinally and transversely, and examined microscopically. It was surrounded by a thick bundle of connective tissue, which sent strong processes into the mass and separated the bundles one from another. These bundles were cut across in every imaginable direction, but superiorly were principally directed longitudinally. They consisted of closely aggregated very small nerve-fibres, composed in every case of a fine axis-cylinder surrounded with a thin layer of myeline. The fibres varied somewhat in diameter, the largest being about twice the size of the smallest, but only about one-

sixth the diameter of a normal fibre (not one of which was to be found). What we may call the large primary nerve-bundles were widely separated by channels containing connective-tissue fibres and nuclei.

Each primary bundle was divided into several secondary bundles, separated by narrower channels. All these channels seemed to be connected, and were probably lymph spaces.

The left sciatic nerve was examined at various points. A section two inches above the neuroma (see Fig. 1) showed, with a low power, a slight increase, both in extent and compactness, of the connective tissue and fat between the bundles. The vessels showed no special change, and the perineurium was normal. In the bundle the lymph paths were wider than normal, separating the bundle into well-marked secondary bundles. The channels contained shreds of connective tissue and nucleated cells. The secondary bundles were also divided by narrower channels into minute groups. Some of these consisted of one or more fairly normal fibres with a little connective tissue around; from the great preponderance of myeline and a somewhat atrophied axis-cylinder they appeared lightly stained. Other groups consisted of a single normal fibre with a darkly-stained mass, and others again (and these very largely predominated) consisted only of a dark mass apparently containing no nerve fibres. Thus a primary bundle appeared to be made up of dark groups with an occasional light group, all being separated by wide channels. With a high power the dark masses were easily seen to consist of very minute medullated fibres, exactly like those in the neuroma. They were separated by a deeply-stained tissue, probably connective in nature. To test whether the fine glistening ring round the axis-cylinder was myeline or not, Weigert's hæmatoxylin stain for myeline was used, when, with very thin sections and good daylight, the blue-stained ring with the yellow axis-cylinder and connective tissue were beautifully shown in each case. On staining other sections with a watery solution of logwood many nuclei were discovered, principally between the minute fibres; most of these undoubtedly belonged to connective-tissue cells, but some may possibly have been nerve nuclei. I estimated after much care that the

FIG. 1.—Transverse section of Left Sciatic Nerve in case of Amputation of Left Thigh, showing numerous small fibres and few large fibres. The spaces between the bundles should be quite clear, and not shaded as in the figures Both are magnified 300 diam. E. S. Reynolds, del.

FIG. 2.—Transverse section of Right Sciatic Nerve in case of Amputation of left Thigh, showing numerous large fibres and few small fibres.

total number of fibres large and small, in one field, was about equal to the number seen in a normal nerve in the same field; it was certainly not less, and could not safely be said to be more.

At a point four inches above the neuroma much the same appearances were seen, but already the number of large fibres was slightly increased. Sections taken at the upper end of the left sciatic showed two distinct forms of primary bundles; a less numerous kind composed almost entirely of large normal fibres with a few patches of small fibres; and a much more numerous kind, just like the section at the end of the nerve, with patches of fine fibres (with a few large ones) separated by wide channels. Thus the only difference between the upper and lower ends is the addition in the former of a number of normal fibres, these being almost certainly distributed to the thigh.

The changes in the left anterior crural nerve were much the same as in the sciatic, but were found in a much less marked degree. This we should expect, as the anterior crural is so largely distributed to the thigh.

The right sciatic (see Fig. 2, p. 505) and anterior crural nerves consisted almost entirely of primary bundles of normal fibres. A few patches of small fibres were, however, found, but in very slight amount as compared with the left side.

The nerve roots on both sides were cut away from the cord, thus being, in the case of the posterior, for central to the spinal ganglia. The right anterior root showed nerve fibres beautifully uniform in size and closely packed together, not a single small fibre being found. The left anterior showed a large predominance of normal fibres, but they were separated into small groups by wide channels and increased connective tissue. In addition, the important fact was noticed that many small medullated fibres were seen mixed up in certain bundles with the large fibres. The right posterior root showed fibres varying in size from large to very small, but the former largely predominated; all were closely packed together. The posterior root showed the most marked changes. The fibres varied much in size, but the small predominated greatly over the large; they were divided into small groups by wide

channels and connective tissue, forming a marked contrast to the closely packed fibres of the roots on the right side.

The left posterior spinal ganglia contained a large number of small fibres, and an increased amount of connective tissue, as compared with the right posterior spinal ganglia.

Conclusions.

The few small fibres of the right sciatic nerve are probably due to the general paralysis. This lesion was described by Dr. Bevan Lewis, in the 'West Riding Reports' for 1875, and he, from a theoretical point of view, considered them to be afferent; this seems to be the case, as I only found them in the posterior nerve-root.

The neuroma was seen to be made up wholly of small fibres. These I consider to be regenerative fibres, growing from the fibres above, and so lucidly described and figured by Ranvier, as occurring at the end of the central stump of a cut nerve.

The numerous small fibres of the sciatic trunk I look upon differently, however. They seem to be fibres simply atrophied in all their constituent parts, without any degeneration. This is due purely to disuse, the connection with the trophic centres preventing any degenerative change. Most of them are undoubtedly sensory, and the change could be traced distinctly through the posterior spinal ganglia right up to the cord. But very many are most certainly motor, as proved by the number of small fibres in the left anterior roots. Theoretically, we can easily see that after amputation, say of the thigh, no afferent impulses from the part removed can travel to the cord, and therefore these fibres are absolutely unused. But it is quite possible for motor impulses from the cord to overflow into the efferent nerves of the stump, even if most of them become blocked at the site of amputation. I only throw this out as a doubtful explanation of the greater number of atrophied sensory fibres.

In my case, as above stated, there was ample proof, from an examination of the nerves and their roots, that both motor and sensory spheres were affected. This being allowed, there is no reason to say that the postero-lateral group in the anterior

horn is sensory in function. It was, moreover, by no means the only group of the anterior horn affected, as the central and antero-lateral groups were both markedly changed, again pointing to an implication of the motor sphere. The idea that the postero-lateral group is sensory is quite contrary to all former anatomical and pathological observations; its function almost certainly being, as pointed out by Ross, to innervate muscles which maintain the erect posture.

The affection of the sensory tracts was accompanied by a diminution in size of the posterior column and horn of the same side. It was curious that no unilateral ascending degeneration was noticed in any part of the cord. In my case no definite lesion of Clarke's columns was noticed, and remembering how the number of cells on each side in any one plane vary in this group, there seems hardly sufficient evidence, from the comparatively slight difference on the two sides in Friedländer and Krause's cases, to assert that it is sensory in function. It must, moreover, be remembered that Ross long ago suggested that the cells of Clarke's column were specially connected with the innervation of the viscera. This idea has been worked out and fairly established by Gaskell,[1] and adopted by Ferrier in his new work.

I thus finally conclude that, after amputations of limbs, the changes which occur may be explained simply, and might have been expected from à priori reasoning. The nerve fibres up to and with their centres in the cord atrophy from pure disuse. Each small fibre in the nerves has arisen from one large fibre, and the shrinking which thus would occur appears to be compensated for by the great widening of the lymph channels, and the slight increase of connective tissue in the small bundles.

The unilateral changes in the brain seem only to occur if the amputation is performed early in life.

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CASES OF INJURY TO THE CERVICAL REGION OF THE SPINAL CORD.

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THE following cases of injury to the spinal cord have been treated in the Manchester Royal Infirmary since January, 1885, and appear to be of sufficient interest to warrant their collective publication. For permission to make use of them I am indebted to the surgeons under whose care they have been admitted. I have also to thank the house-surgeons and dressers of the Hospital for much assistance in taking the notes, but I am especially indebted to the Resident Surgical Officer, Mr. Joseph Collier, who has had exceptional opportunities of observing most of these patients throughout their stay in the wards, and who has most generously placed his private notes at my disposal. Except where otherwise stated, the post-mortem examinations have been made by Dr. Harris, pathologist to the Infirmary. I may add that the cases are in no way selected, all injuries of the cervical region of the cord which we have admitted during the last eighteen months being here included. We have, of course, had cases presenting slight bruises of the back, it may be with somewhat prolonged pain and weakness, but in none of these were there any symptoms of injury to the cord, and they are therefore not included in the present series. We have had no case of "concussion of the spine" in which spinal cord symptoms supervened on a remote injury, a general shake, or a slight local injury, nor have I ever seen such a case in the Manchester Infirmary.

The cases arrange themselves into two great classes, the one where the diagnosis was confirmed and the symptoms explained by the death of the patient and a post-mortem examination;

the other where there was fortunately no such demonstration of the pathological condition. It will be convenient to consider first the cases in the former group, and then to endeavour, by the light thus gained, to realise more fully the significance of the remainder.

CASE I.—*Fracture-dislocation between the Fifth and Sixth Cervical Vertebræ.—Complete Paralysis of all Nerves below the Fifth Cervical.—Death.*

L. F. was admitted to the Manchester Royal Infirmary under the care of Mr. Jones on June 5th, 1886. The patient was a man, aged 68, a labourer by occupation. On June 1st, four days before admission, he was standing on a short ladder whitewashing, when, two of the rungs of the ladder giving way, he fell backwards in such a position that while his feet remained caught in the ladder, he first struck the back of his neck against a bench some two-and-a-half feet above the ground, and his head then fell to the ground. At the same time he thinks that his bucket of whitewash fell upon him.

He at once became unconscious, remaining so for several hours. On regaining consciousness he found that his legs were quite immovable and his arms partially so, while there was numbness in the lower portion of his body, and insensitiveness to external impressions, coupled with a dull aching pain. There was also great pain in the head, neck, and shoulders. The urine had to be drawn off twice daily. He remained in this condition until his admission to the Infirmary on the fourth day after the accident.

When admitted he was found to present complete paralysis of the lower limbs, and of the abdominal and thoracic muscles. All the muscles of the arms were paralysed, with the exception of the biceps, brachialis anticus, supinator longus and deltoid, the consequence being that the elbows were flexed, the shoulder abducted and rotated outwards, and the hands and arms fell into the position indicated in the annexed engraving (Fig. 1, p. 512), taken from a photograph for which I am indebted to Mr. Sidebottom. There was no power of extension of the forearm, but a fair degree of power of flexion. Some rotation of the humerus could be effected, apparently by the fibres of the deltoid. The contractility of the supra- and infra-spinati was not ascertained; the pectoralis major and latissimus dorsi were paralysed. The lower limbs and trunk were completely anæsthetic as high as

the second rib in front,—*i.e.* as high as the descending branches of the cervical plexus—and apparently to the third dorsal vertebra behind. Above this point sensation was normal. The upper extremities were anæsthetic, with the exception of the outer side of thefore arm and hand and the balls of the thumbs. Both cutaneous reflexes and tendon reactions were absent throughout. Breathing was entirely diaphragmatic. There was retention of urine, and the bowels had not been moved since the accident. The penis was subject to frequent erections with seminal emissions; on the back were several bed-sores. The pulse was slow and full; the

Fig. 1.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the level of origin of the fifth cervical nerves.

temperature 98°. The pupils were both somewhat contracted. Over the 5th and 6th cervical vertebræ was a slight prominence, which was very tender on pressure. The only treatment adopted was complete rest on a water bed, catheterisation at intervals, and some simple dressing for the bed-sores.

During the progress of the case there were not many very important deviations from the symptoms above described. The urine, which was at first healthy, became turbid and alkaline a few days after admission. After this the bladder was washed out daily with boracic lotion. Priapism became more constant. The bowels were never moved except after the use of an enema, or of croton oil. On June 9th, four days after admission, the arms had become completely anæsthetic and remained so throughout. The temperature was generally slightly below the normal; the pulse

was often 50, never above 60. Respiration was rapid throughout, being usually about 20, sometimes 30 per minute.

The patient's strength failed steadily, and at times there was confusion of thought, with low muttering delirium at night. He complained mainly of pain in the neck, and of cold feet.

On June 15th he had lost all power of rotating the humerus, on both sides, and passive movement of the shoulder-joints was very painful. The cheeks fell in and out very markedly on respiration. Although the power of the voice remained good, speech was almost unintelligible; he could not protrude the tongue beyond the teeth, nor freely open the mouth. There was marked fibrillar trembling of the masseters; and much accumulation of tenacious saliva about the mouth. There were bed-sores over the sacrum, on the inner side of the right thigh, and on the inner side of the left knee, these having hardly changed since admission.

On June 17th the contraction of the deltoid muscles, which had maintained the abducted position of the arms, ceased, and the elbows fell to the sides, the elbow-joints themselves remaining flexed.

From this date the patient gradually sank, respiration becoming more laboured, and the countenance dusky, while there was increasing apathy. On June 25th the pupils were noted as being dilated and unequal, the right being the larger; there was a commencing "bed-sore" on the chest; on washing out the bladder there came away shreds of what was thought to be mucous membrane. On June 26th he died exhausted.

At the *post-mortem* examination was found depression of the 6th cervical spine and laminæ; the disc between the 5th and 6th vertebræ was broken across, the upper part of the body of the 6th vertebra being tilted backwards. The laminæ were uninjured, but the right superior articular process of the 5th vertebra, and the corresponding transverse process of the 6th were broken off. At this point the cord was compressed, being flattened for a distance of about $\frac{1}{4}$ inch. The dura mater was here yellowish and opaque, the arachnoid and pia mater healthy. The cord was much softened for a distance of two or three inches above and one inch below the seat of injury, especially around the central canal, where it was quite diffluent and of a yellowish colour, showing under the microscope numerous granule cells. The rest of the cord was healthy. The bladder contained a quantity of blood-stained turbid fluid, its mucous membrane hanging in shreds, beneath which the wall was dark and livid; inflammation had extended to the pelvic cellular tissue and

peritoneum. The kidneys were congested, and presented numerous scattered points of suppuration; there was no dilatation of or suppuration in their pelves. The other organs presented nothing of interest.

This case is a sufficiently typical one of injury to the spinal cord above the origin of the 6th cervical nerves. The most interesting feature presented by it is the distribution of the paralysis and anæsthesia. Thus we find that, with the exception of the deltoid, biceps, brachialis anticus and supinators, there was absolute paralysis of the muscles of the upper extremity. The case is thus an exact counterpart of the so-called Erb's paralysis, in which we meet with paralysis of the above named muscles only, and enables us to confirm previous researches as to the distribution of the 5th cervical nerve-root. Besides the intrinsic muscles of the arm there was certainly paralysis of the pectoralis major, which is supplied by the external and internal anterior thoracic nerves, and presumably also of the pectoralis minor. The latissimus dorsi appeared to be paralysed, and no doubt it had lost its nerve supply, from the long subscapular, as well as from the numerous smaller dorsal and lumbar branches, far below the seat of the lesion. That the subscapularis was paralysed appeared to be indicated by the external rotation of the humerus, in marked contrast to the internal rotation seen in Erb's paralysis and in obstetrical paralysis, where this muscle is spared. It was not found practicable to ascertain the condition of the teres major and minor, but the fact that the former is supplied by the middle subscapular would render it probable that it was paralysed with the subscapularis and latissimus, whereas the nerve to the teres minor is the circumflex, and we would therefore expect it to escape with the deltoid. The teres minor would also aid the posterior fibres of the deltoid in producing the well-marked external rotation of the humerus. The serratus magnus, supra- and infra-spinati remain for consideration; but on the innervation of these three muscles we have here no information.

In Erb's paralysis these muscles escape, and would therefore not appear to be supplied by the 5th root; in the closely-allied form of peripheral lesion described by Duchenne as "obstetrical paralysis," the spinati muscles are affected, and it would hence

appear that the 4th cervical root supplies them with nerve fibres. Certain it is that they must be supplied by either the 4th, 5th, or 6th cervical, inasmuch as their nerve, the suprascapular, is derived from the cord formed by these three, before its junction with any other root, and as the 6th root is found to supply the triceps, which is not affected in either Erb's paralysis, or obstetrical paralysis, we must refer the innervation of these muscles to the 4th. Thus, then, we arrive at the result, that the 5th cervical nerve supplies only the deltoid, biceps, brachialis anticus and supinator longus (with probably the supinator brevis and teres minor also).

As regards sensation, we find that a cord lesion which cuts off the whole of the nerves of the brachial plexus below the 5th cervical causes anæsthesia of the whole of the upper limb, except the outer side of the arm and forearm, and the radial border of the thumb, *i.e.*, a part of the region supplied by branches of the musculo-spiral nerve. Possibly the area in which sensation was retained was at first more extensive than the above, inasmuch as the case was only seen for the first time four days after the injury, and we find that by the 8th day all sensation was lost in the upper extremity: a change due, no doubt, to extension of myelitis from the seat of injury. To this same zone of myelitis we must ascribe the contracted condition of the muscles supplied by the 5th cervical root, a contraction which slowly gave way to paralysis, as irritation was followed by destruction of the grey matter of the cord.

The affection of speech and other symptoms noted in the progress of the above case, at first gave rise to the supposition, that there might be some extension of myelitis to the medulla oblongata; but as no further symptoms of bulbar paralysis developed themselves, and as the intermediate cervical muscles were unaffected, these phenomena would appear to be due to the general debility only.

CASE II.—*Fracture-dislocation between the Fourth and Fifth Cervical Vertebræ.—Complete Paralysis of Limbs and Trunk.—Death.*

The following case was under the care of Mr. Heath. The notes were taken by the dresser, Mr. W. H. Iddon.

M. L., male, aged 45, was admitted on March 27th, 1886. He

had fallen from a scaffolding some forty feet in height. In addition to three lacerated scalp-wounds, he presented the following symptoms. There was severe pain in the back of the neck. All four extremities were completely paralysed, as were the muscles of the trunk and abdomen. The limbs and trunk were absolutely anæsthetic below a line running across the thorax at the level of the second intercostal space, and thence across the deltoids at the junction of their upper and middle thirds; above this line sensation was normal, the transition being quite abrupt. The pupils were equal, and appeared to be of normal size. There was no mental affection. Respiration was diaphragmatic; the urine was retained; priapism was constant. Death took place a few hours after admission, apparently from dyspnœa.

The temperature was taken several times, but was unfortunately not recorded. The house surgeon assures me that it was certainly not, at any time, far from the normal.

At the *post-mortem* examination there was found a fracture through the body of the 5th cervical vertebra, and the intervertebral disc immediately above it, The spines of the 3rd, 4th and 5th vertebræ were broken off. Outside the dura mater at this point was a very slight effusion of blood. The cord was much lacerated, being almost torn across opposite the seat of fracture, and was very soft for a distance of nearly 1½ in., being reddened by extravasated blood. The skull and its contents were uninjured. The lungs were œdematous.

The above case contrasts well with Case I. Here we have the lesion affecting the 5th vertebra and the disc immediately above it, so that the 5th root, which escapes from the spinal canal above the 5th cervical vertebra, would be injured, as well as all parts below. Hence we get complete paralysis of sensation and motion in the upper extremity, the whole of the brachial plexus being cut off from the brain, with the exception of the branch of the 4th cervical nerve. The present cases unfortunately yield no evidence as to the distribution of this branch, the condition of the posterior scapular muscles not having been specially noted.

CASE III.—*Dislocation between the Fourth and Fifth Cervical Vertebræ. —Complete Paralysis of Limbs and Trunk.— Death.*

C. D. was admitted under the care of Mr. Hardie. The notes were taken by Mr. Benson, House-surgeon. The patient was a

carter, 39 years of age. He was admitted on August 3rd, 1886, having fallen from a van, of which the wheels had passed over his shoulders.

On admission he complained of great pain at the back of the neck. All four limbs were completely paralysed, and there was anæsthesia below the level of the descending branches of the cervical plexus. Respiration was diaphragmatic. The fæces were passed unconsciously. Both pupils were contracted, the palpebral fissures narrowed, and the eyeballs presented a remarkable sensation of softness owing to paralysis of the posterior orbital muscles. Pulse was full, of low tension, and beating at the rate of 60 per minute. The temperature on admission, which was in the evening, was 96·8°.

He passed a restless night, and on the next morning the following additional symptoms were noted. There was constant priapism. The urine was retained, and great difficulty was found in passing a catheter, due apparently to the extreme dryness of the urethra, as it entered readily after an injection of oil. The urine was examined for sugar and albumin, with negative results. The face was much congested; the pulse remained full and soft, beating at the rate of 130 per minute. The temperature at 8 A.M. was 102·2°. An interesting fact noted by Mr. Collier was, that when the patient was told to take a deep breath, he produced contraction of the levator anguli scapulæ muscle, which could be distinctly felt beneath the trapezius, thus indicating an attempt to fix the scapula, and bring into play the extraordinary muscles of inspiration.

At 10 A.M. the patient's temperature had risen to 103·6°; at midday to 105°, at 2 P.M. to 106°. At 4 P.M. it was 107·8°. He now became insensible and livid, and the breathing more shallow. The whole body was covered with a profuse hot sweat. At 5 P.M. he died, the temperature having risen to 108°. The temperature after death was not recorded.

At the *post-mortem* examination there was found a rupture of the cartilage between the bodies of the 4th and 5th cervical vertebræ, these two bodies being so widely separated behind that a finger could be introduced between them. They thus formed an obtuse angle posteriorly, pressing on the contents of the spinal canal. At this point there was marked post-mortem staining of the meninges, but decomposition was so far advanced that it could not be ascertained whether they were inflamed. The cord itself was here compressed, through a vertical extent of $\frac{3}{4}$ of an inch, into a narrow band, and was very much softened, there being no distinction between the grey and the white matter. Elsewhere the cord showed only post-mortem softening.

The posterior parts of both lungs were much congested, and in the brain were numerous puncta cruenta. There were no other changes of interest.

This case is similar to the last, the lesion again involving the 5th cervical nerve and all parts below it, and the symptoms being the same. We shall refer again to such further points of interest as it presents.

CASE IV.—*Fracture-dislocation between the Fourth and Fifth Cervical Vertebræ.—Partial Paralysis of Right Upper Limb.—Total Paralysis of other Limbs and Trunk.—Death.*

T. L. was admitted under the care of Mr. Heath on Sept. 7th, 1886. He was 36 years of age, and a carter by occupation. While attending to his horse the animal had fallen against him, crushing him against a manger, the corner of which struck his back between the shoulders. He immediately fell down paralysed.

On admission there was found no external bruise or other evidence of injury, except a sense of soreness and stiffness in the lower cervical spine; no irregularity of the spinous processes could be detected. The lower limbs and trunk muscles were entirely paralysed, as was the left upper extremity; the right upper extremity was paralysed with the exception of the deltoid and biceps (the condition of the brachialis anticus and supinator longus is not noted). The bladder and sphincter ani were also paralysed, and there was priapism.

There was anæsthesia of the lower limbs and trunk, and of the upper limbs with the exception of the back of the hand, forearm and arm on both sides. Breathing was entirely diaphragmatic. The pupils were widely dilated. The temperature was 100·6°. The treatment consisted in the use of a water bed with ice-tubes to the spine, doses of ext. ergot. liq., three times daily, and catheterisation.

At 8 A.M. on the following day the temperature had risen to 105·2°; at noon it was 104°; and then again gradually rose, reaching 105·6° before death, which occurred on the second day. Breathing became more difficult, with accumulation of mucus in the bronchial tubes, and the face grew very livid. He died asphyxiated about thirty-six hours after admission, the heart continuing to beat for some ten minutes after respiration had stopped. An unsuccessful attempt was made to save the patient by bleeding from the arm, artificial respiration, and stimulation with the faradic current.

At the *post-mortem* examination there was found a rupture of the cartilage between the 4th and 5th cervical vertebræ, "which fracture had extended across posteriorly, involving the laminæ of the 4th or 5th vertebræ." The spinal membranes were healthy. The cord opposite the seat of fracture was not at all compressed, but was for a distance of about an inch very soft and pulpy, containing numerous punctiform hæmorrhages, which were most marked in the central grey matter. The other organs were not examined.

This case presents an interesting comparison with Case I. on the one hand, and with Cases II. and III. on the other. The lesion of the spine itself was less severe, and there was less displacement of the bones, which consequently did not cause permanent compression of the cord. There was, however, ample evidence of a temporary crush of the spine, received, no doubt, at the time of injury, the bones afterwards recoiling partially to their normal position. Such, probably, is the explanation of many cases (as, for instance, our sixth case), where there is little or no evidence of bone lesion, but the cord itself has sustained a severe injury. An accident causes an acute bend of the vertebral column, which at once rights itself, but not before great or irreparable damage has been inflicted upon the contained organ; and many of these cases have been regarded as instances of "concussion of the spinal cord," the possibility of a gross mechanical lesion being overlooked. Another point which is here exemplified, but which is more fully demonstrated in Case VI., is the tendency for hæmorrhage into the spinal cord to affect mainly its central portions, where its substance is softest and where the large branches of the central spinal artery have their distribution.

As regards the distribution of the paralysis and anæsthesia in this case, we find that there was not complete annihilation of the functions of the two 5th cervical nerves. On the left side, the upper limb was completely paralysed, as in Cases II. and III., but on the right side the muscles supplied by the upper root of the brachial plexus had partially escaped, the biceps and deltoid not being paralysed. As regards the supinators and the brachialis anticus we have no information, and I did not see the case, but should suppose that these also

probably escaped, although it is quite conceivable that their spinal motor cells may lie a little lower in the cord and may thus have been involved in the lesion. Further, we find that as in Case I., so here, sensation was retained in the distribution of branches of the musculo-spiral nerve, on the outer border of the arm, forearm, and hand of both sides.

CASE V.—*Comminuted Fracture of Fifth and Sixth Cervical Vertebræ. —Partial Paralysis of Left Upper Limb.—Total Paralysis of other Limbs and Trunk.—Death.*

W. H. was admitted to Mr. Whitehead's wards on April 30th, 1886. He was 28 years of age, a wine-merchant by occupation, and of intemperate habits. When admitted he was intoxicated, and had, while in that condition, fallen over the bannisters of a staircase, the distance not being ascertained. He complained of great pain at the back of the neck; but there were no external signs of injury. There was complete paralysis of both lower extremities and of the right upper limb; but on the left side he could bend the elbow-joint. Respiration was diaphragmatic. The cutaneous reflexes and tendon reactions were abolished. Anæsthesia was complete below the branches of the cervical plexus, except over a portion of the left upper limb. The pupils were moderately contracted, the contraction—or rather imperfect dilatation—showing best, as is usual in these cases, in diffused light: the palpebral fissures were small. The pulse was slow and compressible. The temperature was not recorded, but was below the normal. The urine was retained, and the penis large and turgid.

A more complete examination showed, that while there was absolute loss of motion and sensation in the right upper extremity, the paralysis had on the left side spared the deltoid, biceps, brachialis anticus and supinator longus; the pectoralis major and latissimus dorsi, and all the muscles below the shoulder, with the exception of those mentioned, were paralysed. The arm occupied exactly the position assumed by those of Case I., being slightly abducted at the shoulder, rotated outwards, with the elbow flexed and the forearm and hand supine, the contrast between this position and the complete flaccidity of the right upper extremity being very striking. This was the first case in which this remarkable position of the limb was noted in the Manchester Infirmary, the detection of its significance being due to Mr. Collier. Its importance is shown by the fact, that it was found possible to ascertain the exact nature of the cord lesion before death. Anæsthesia

extended over the whole of the left upper extremity, except a strip of skin, some three inches wide, extending downwards from the shoulder, along the outer side of the limb to a point about three inches below the level of the elbow-joint.

The patient complained much of difficulty of breathing, which increased rapidly, and he died asphyxiated about forty hours after the accident. Before death the temperature became very high.

At the *post-mortem* examination, which was made by Dr. Bury, Medical Registrar, there was found a complete crush of the body of the 5th cervical vertebra, the 4th being displaced downwards and forwards, with its left transverse and articular process broken, and

FIG. 2.—Third, fourth, fifth, sixth and seventh cervical vertebræ from Case V., seen from the front, showing a comminuted fracture of the bodies of the fifth and sixth vertebræ.

the 6th split vertically: the condition of the bones is shown in Fig. 2. In front of the bodies of the injured vertebræ was some extravasation of blood. On opening the spinal canal, the cord was found to be obliquely compressed in such a way that the upper limit of the flattening was above the 5th cervical nerve on the right side, but between that and the 6th nerve in the left, as shown in Fig. 3 (p. 522), a small piece of the body of the fifth vertebra pressing upon the 5th root of the right side. The lungs were intensely congested, and portions of the left lower lobe sank when thrown into water. The other organs presented nothing of interest.

This case, like the preceding one, is an instance of oblique

injury to the cord, the direction of the obliquity being reversed, and the 5th root escaping on the left side only; and it thus offers another illustration of the distribution of the motor and sensory branches of this root. The area in which sensation was retained was less than in Cases I. and IV., owing probably to some of the lower fibres of the left 5th root also being involved in the lesion.

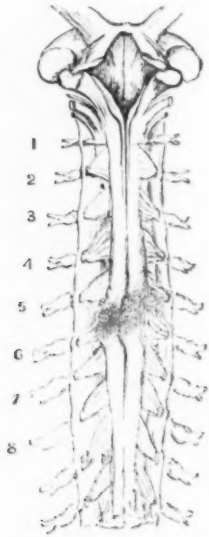

FIG. 3.—Spinal cord from Case V., seen from behind, showing compression, extending obliquely from above the origin of the sixth cervical nerve on the left side, upwards, so as to intercept the fifth nerve on the right side.

The above have been cases of fracture of the cervical spine, but the next on our list differs in that the lesion was not a fracture, but a traumatic hæmatomyelia, there being no obvious lesion of the spinal column itself.

CASE VI.—*Traumatic Hæmatomyelia in Lower Cervical and Upper Dorsal Region.—Death.*

J. B., aged 34 years, was admitted on December 30th, 1885, under the care of Mr. Heath, suffering from an injury to the spine. He was a carter by occupation, and was hurt, while loading a

waggon, by a "tippler" full of coal falling upon him, so as to throw him upon his face, the coal striking him between the shoulders.

On admission to the Infirmary he was quite conscious, but stated that he had been rendered insensible for some time by the injury. He had complete loss of power over both lower extremities, and complained of great pain at the back of the neck and between the shoulders, which was increased by moving the head. He had also pain from a severe bruise over the right elbow. On the left side of the head were three slight scalp-wounds.

A more careful examination showed him to have pain mainly over the 5th and 6th cervical vertebræ, increased by pressure; but no irregularity was here detected in the spine. There was absolute paralysis of both lower extremities, with deficient action of the intercostal and anterior abdominal muscles in respiration, but good movement of the diaphragm. Both legs were completely anæsthetic as high as the knees; but thence upwards he had some sensation, although there was distinct numbness as high as a line drawn round the abdomen about two inches below the umbilicus. There was no hyperæsthesia. The plantar reflexes were noted as "almost absent," and the feet felt very cold, with some prickling sensation. He had distinct priapism. Both pupils were much contracted, but equal and contracting further on exposure to light. There was no vomiting and no mental affection. He was placed on a water bed, the scalp-wounds dressed, and ext. ergot. liq. administered in ʒss doses. In the evening the urine had to be drawn off.

On the following day there was still absolute paralysis of the lower limbs; but there was now no anæsthesia. The priapism had subsided, and the urine was passed without any trouble. The diaphragmatic respiration was very marked. On examining the arms, which had not been done the day before, I found distinct feebleness of the flexors of the wrist and fingers on both sides; the grip was very feeble, but the power of extension against resistance little if at all diminished. Both superficial reflexes and tendon reactions were everywhere absent. The left pupil was somewhat contracted, but the right one very much more so; both contracted further on exposure to light. The patient had some vomiting during the night, and there were râles all over the chest. The morning temperature was 98·6° F.; in the evening it was 99·8° F.

On the following day, January 1st, 1886, his fæces were passed involuntarily. There was severe and painful cough, and all over the chest loud crackling râles; the countenance was cyanotic.

No other change had occurred. Temp., morning, 99°; evening, 100°. The cyanosis and difficulty of breathing now rapidly increased, and there was dulness at the bases of both lungs, but no crepitations were heard. Without further symptoms, the man died on the night of January 2nd, 1886.

At the *post-mortem* examination there was found engorgement of the bases of both lungs and of the right side of the heart; but the other organs were healthy, with the exception of the spinal region, which is thus described. "A small dark black hæmorrhagic effusion existed in the muscles over the lower cervical and upper dorsal regions." The membranes of the cord were quite normal, and the external appearance of the cord itself presented no evidence of injury; but "on section there was found to be a dark black hæmorrhage into the central grey matter, in the lower cervical and upper dorsal regions. This hæmorrhage, which measured in its vertical extent from 1½ to 2 ins., was in the greater part of its extent situated centrally, occupying the whole of the central grey matter and extending but little into the white substance, which in its neighbourhood was merely softened and of a faintly yellow tinge. At the lower part, for a very short distance, the hæmorrhage was limited to the anterior cornu of the right side, while the corresponding left horn appeared to be perfectly healthy. Elsewhere the cord was firm, and presented no abnormality."

The above case presents a large number of points of interest. In the first place, as regards the pathology, we see here, as in Case IV., the tendency for hæmorrhages in the cord to affect the region immediately surrounding the central canal, where the vascular supply is very free. But the question which at once presents itself is, how are we to explain the occurrence of this hæmorrhage without an injury to the vertebral column? It appears to us impossible to suppose that it was due solely to concussion—to an indirectly-transmitted shake or jerk of the cord. If such were the method of its production, and if it were due to the cord being jerked forwards against the bodies of the vertebræ, we should expect the post-mortem evidence of the bruise, thus given to that delicate structure, to extend over a wider area: the cord would, in such an injury, be thrown forwards without any acute bend, and we should expect to find, not a single localised hæmorrhage, but a more diffused and extensive lesion. It appears far more probable that the mode

of production of the injury was a sharp bend of the vertebral column at the region where it was struck—the blow being, as we have seen, a direct and severe one—the bones at once recoiling, but the more delicate cord being partially ruptured at the point of this sharp bend. A condition somewhat similar to this is seen in Case IV., where, however, the spinal column shows the effects of the strain to which it has been submitted, although it has recoiled sufficiently to obviate permanent compression of the cord. In the present instance we have only to suppose the injury to the framework to have been a little less severe than in Case IV., and the crush to its contained viscus to have been practically the same. This explanation of a temporary bend of the spine, with consequent injury to the cord, is advanced by Mr. Page in the July number of 'Brain,' as the true pathology of MM. Duménil and Petit's case of "concussion" there described; and we would suggest that the present case not only confirms Mr. Page's view, but that the case which he quotes was probably of exactly the same nature as the one at present under consideration. Had our case survived for thirty-five days, as did that of MM. Duménil and Petit, we should have expected to find exactly the appearances described by them, viz. a patch of softening and discoloration at the point struck, with probably a secondary degeneration of the lateral columns below that region.

If, however, we are to assume the present case to be one of direct injury and not of "concussion" of the cord, it will be necessary for us to explain the various symptoms produced by this comparatively slight lesion, and this there appears to be no difficulty in doing. It is well recognised that in the case of cerebral hæmorrhage, we meet with two classes of symptoms, viz. those due to the actual destruction of a portion of the brain substance by the effused blood, which are permanent; and those due to compression of surrounding tracts or nuclei, which are more or less evanescent, their disappearance being conditional on the absorption of the blood and consequent diminution of the compressing force. Thus it may be and often is the case, that the "compression symptoms" entirely overshadow the "destruction symptoms," and that a

comparatively trifling hæmorrhage may cause extensive and more or less persistent compression of important portions of the brain. Precisely similar appears to have been the condition in the present case. The hæmorrhage itself occupied only the region of the central canal in the lower cervical and upper dorsal portions of the cord, extending to but a slight extent into the right anterior cornu. But this effusion of blood would suffice to cause more or less pressure upon the whole of the cord at this level, a pressure to which probably that structure would soon accommodate itself, having, as it has, ample room to expand. Perhaps then it will not be too bold an assumption to suppose that the direct symptoms, due to destruction of the grey matter by the blood, were insignificant; the other and more serious results of the injury arising from mere compression by this effusion.

There is no difficulty in attributing the paralysis of the lower part of the body to compression of the descending columns of the cord. The anæsthesia was partial only, becoming somewhat gradually less marked towards its upper limit, and this would coincide with the view, that the sensory fibres for the lower part of the body are more centrally situated in the spinal cord: a condition obviously convenient in view of the fact, that they have a longer course to travel. If this be their arrangement, we should expect exactly the condition which we find, viz. a more severe compression, and consequently more absolute loss of conduction of the deeper fibres, those most superficially situated having apparently escaped altogether. Again, in the case of the arm muscles, we find that the extensor muscles of the wrist and fingers have apparently escaped entirely, whereas the flexors are partially paralysed. Now, the hæmorrhage, extending as it did to the lower cervical region, would compress the anterior cornua in that region, and it is well recognised that it is from this portion of the cord that the flexors of the wrist receive their nerve supply: these muscles are not completely paralysed, as would be the case in a destroying lesion of their nuclei, but partially so, as we should expect in a compression lesion. On the other hand, the extensors of the wrist and fingers derive their nerve supply from a somewhat higher point in the

cord, probably from the 6th or 7th cervical nerve, and these muscles are little, if at all, affected, while the muscles, already so often mentioned, which are supplied by the 5th cervical root, certainly escaped entirely. Finally, the early diminution of the anæsthesia, and of other symptoms such as the priapism and retention of urine, would well accord with the theory of their production by a compression of the conducting fibres, to which the latter rapidly accommodate themselves. On these grounds then we hold, that in no sense can the above case be regarded as one of concussion of the cord, but rather as one of a direct blow causing a small local hæmorrhage, with important symptoms arising from the compression thereby induced.

We now leave the region of diagnosis by post-mortem examination, and pass on to the consideration of some cases of recovery after injury to the cervical spine.

CASE VII.—*Traumatic Hæmatomyelia in Middle Cervical Region. —Recovery with Persistent Symptoms.*

W. H., aged 36, a carter, was admitted under the care of Mr. Hardie on June 13th, 1885. About half-an-hour before admission he had fallen between the shafts of a van which he was driving, one wheel of which had passed over his shoulders. It may be remarked that there was no history of syphilis.

On admission he lay quite helpless, there being complete paralysis of the left arm and of both legs, while the right arm was partially paralysed, but retained some motor power "especially at the elbow." Respiration was diaphragmatic. The reflexes were normal. There was anæsthesia below the distribution of the cervical nerves. Speech was laboured, owing to difficulty in breathing, but there was no loss of consciousness or mental confusion. The left pupil and palpebral fissure were slightly smaller than those of the right side. There was much pain in the back of the neck, but no abnormality was there detected. After he had been put to bed, he was able to move the right lower extremity slightly. He was placed on a water bed, the head being fixed by sand-bags. There was great thirst and dryness of the mouth.

The urine had to be drawn off; it was acid, of a specific gravity of 1010, and contained neither sugar nor albumin. He passed a fairly good night.

On the following morning it was ascertained that sensation was nowhere absolutely lost, but that it was impaired on both sides of

the body, but more markedly on the right than on the left. There was more power of moving the right leg and arm; the limbs of the left side remained completely paralysed. Other symptoms continued as before; the temperature was, in the morning, 100°2 in the evening, 101°. There was free perspiration, the urine was scanty and high-coloured.

When examined on June 15th, forty-eight hours after the accident, he was feeling much better and had less pain in the neck. He had more power over the right arm and leg, and an examination of the arm showed that the rhomboids, posterior scapular muscles, deltoid, biceps and supinator longus acted well, whereas the other muscles of this limb were paralysed. The left arm and leg remained completely paralysed. Sensation was returning on both sides, but remained less acute on the right than on the left side. This diminution of sensation extended as high as the supra-clavicular branches of the cervical plexus. The reflexes were normal. There appeared to be some slight movement of the intercostal muscles in respiration. The pulse was beating at the rate of 102; the temperature was, in the morning 102°; in the evening, 102°·4.

On the following day there was further gain of sensation and of power on the right side. On the left side were slight pricking sensations. The temperature had fallen to 99°·4. Other symptoms were as before. It was noted that there had throughout been no priapism.

On June 17th he passed his urine without the use of a catheter, and had no further trouble with it. His bowels were now opened for the first time since the accident. The pulse remained somewhat rapid, being 85 in the morning and 100 in the evening; the temperature was 101°. On the 19th there was distinct costal respiratory movement. The pulse was still rapid and the temperature 100°. On the 26th the temperature for the first time reached the normal, at which it afterwards remained, except temporarily on two unexplained occasions.

On June 24th there seemed to be little or no further change. He was ordered iodide of potassium in gr. 10 doses. From this point improvement progressed very slowly. On July 4th he was able to move his shoulders slightly on both sides, and the intercostal muscles acted more freely on the right than on the left side; there was no other change in the paralysis. The knee-jerk was exaggerated on both sides; there was no ankle-clonus. On July 20th he was transferred to the medical wards under the care of Dr. Ross, where very full notes of his condition were taken by Dr. Bury, Medical Registrar, of which an abstract only is here

given. At this time it was noted that costal respiration was still deficient, being apparently more so in the lower ribs (probably because the downward traction of the diaphragm was able to overcome the expansion).

The following was the condition of the right upper limb:—The wrist was semiflexed and somewhat inclined towards the ulnar side, having very slight power of flexion and extension. The thumb lay fully extended, but could be slightly flexed, adducted and abducted: there being, however, no power of opposition. The first phalanges of the fingers were extended, the second and third partially flexed; there was slight power of flexion of the metacarpo-phalangeal joints, but no movement at the inter-phalangeal joints. As regards lateral movement there was slight power of abduction and adduction of the first and fourth fingers, but the second and third were immovable, having always a slight interval between them. He could pronate the wrist and then bring it half-way into the supine position, but not beyond this. The elbow was semiflexed and could be partially flexed, but not extended and passive extension caused the tendon of the biceps to stand out very sharply. At the shoulder-joint there was diminished movement, especially of abduction, external rotation and flexion; he could touch the tip of the opposite shoulder but not his head. There was marked wasting of the whole limb. A very powerful faradic current caused slight contraction of the extensors and flexors of the elbow, and of the serratus magnus, but of none of the other muscles of the limb or shoulder. The galvanic current gave the "reaction of degeneration" with the pectoralis major, deltoid, supra- and infra-spinatus, biceps, triceps, extensors and flexors of the forearm and muscles of the hypothenar and thenar eminences. On the left side the condition was similar, except that there was even less power of voluntary movement. In brief, there was well-marked "atrophic paralysis" of all muscles supplied by the brachial plexus. The movements of the lower limbs were weak, but of normal range, except for some deficiency at the left ankle, and the electric reactions were also here normal.

It was not easy to map out fully the extent of sensory deficiency, owing to the fact that there was nowhere absolute anæsthesia, nor any defined limit. On both sides sensation was normal in the neck, and as far downwards as the lower limit supplied by the cervical plexus, that is, to the level of the third rib. There was below this limit marked diminution of sensation on the right side extending to within ½ inch of the middle line in front: at this point there was an area of transition, and to the left of the middle line sensation was normal. Behind, the anæsthetic area extended over the whole of the right side of the trunk as far as a

vertical line, midway between the posterior border of the right scapula, and the spines of the vertebræ; the upper limit of anæsthesia being here the spine of the scapula. The right shoulder was not anæsthetic. There was no anæsthesia of the left half of the trunk. The right lower limb was anæsthetic, the left was not—in fact, there was partial anæsthesia of the whole of the right side of the trunk and lower extremity, with normal sensation on the left side. In the upper limbs the distribution of anæsthesia was symmetrical, affecting the whole of the limbs, excepting a strip of skin, running from the shoulder, along the centre of the biceps, and then straight downwards along the front of the forearm to the hand, where it spread out, so that the only part of the hand which was anæsthetic was the thumb, the rest having apparently normal sensation both in front and behind.

The knee-jerk was much exaggerated, and there was marked ankle-clonus; the superficial reflexes were absent, and there were no reflexes in the upper limbs.

The skin over the anæsthetic areas was hot and dry, the slightest prick or scratch causing a bright red spot or line. There was slight swelling and tenderness at the back of each wrist. The inter-phalangeal joints were also thought to be slightly swollen, but were probably merely rendered more prominent by the general atrophy. The finger-nails were long, the skin at their roots being red, smooth, and shining.

The left pupil was slightly smaller than the right, and the left palpebral fissure somewhat narrowed.

The patient remained under observation for rather more than three months after this, taking a mixture of iron and quassia. During this time voluntary power increased considerably in the right upper extremity, and to a slight extent in the left; when last seen he could move the fingers of the right hand fairly well in all directions, had slight power of opposition of the thumb, could raise the elbow to the level of the shoulder, and could touch the top of the head with his hand. Sensation had also improved. There was still some difficulty in moving the left ankle, but otherwise the movements of the lower limbs were good.

Superficial reflexes were obtained over the abdomen; the cremasteric reflexes were still absent, the plantar was got on the right side only. The knee-jerk was less marked on the right side than it had been, but was unaltered on the left. There was no other change, and the vital functions were well performed.

Regarded in the light of the preceding cases, there can be little difficulty in following the course of events in the present

instance. The primary lesion was probably a hæmorrhage into the cord situated similarly to that of Case VI., but of somewhat greater extent. On the left side it appears to have extended sufficiently high to annihilate, either by pressure on or by destruction of the grey matter, the whole of the functions of the nuclei connected with the brachial plexus of that side. On the right side the nuclei of the 5th cervical nerve have escaped, as have the pupillary fibres. The parts of the cord below this point have also been affected, probably owing to compression of the conducting tracts, and thus we find partial anæsthesia, which undergoes gradual improvement; and retention of urine, which soon passes off. It is noticeable that this is the only one of our cases of cervical injury in which there was no priapism, and that in Case VI., which was also an instance of mere temporary compression of the conducting paths, priapism was of short duration. In the present instance, as in Case VI., there is paralysis of the lower part of the body, owing to pressure on the lateral columns, and at a later stage we have clearly developed the symptoms of descending degeneration of those columns. The simplicity of the case is, however, interfered with apparently by a localised myelitis arising round the hæmorrhage and thus causing some extension of its destructive effects. Hence in the later stages of the case we find the 5th cervical nerve losing its functions on the right as well as on the left side. That the left side was the more severely injured is evidenced by the fact, that the anæsthesia was more complete and more permanent on the right side of the body. It will also not escape notice that, whereas the muscles supplied by the brachial plexus presented the "reaction of degeneration," those of the rest of the body did not, thus evidencing farther a "destroying lesion" of the grey matter of the cord in the lower cervical region on the one hand, and a mere interference with conduction to the parts below on the other. In the same way we find that in the case of the brachial plexus, sensation is impaired on both sides, whereas, below this point, the anæsthesia is limited to the right half of the body, corresponding with the obviously greater extent of the lesion on the left side of the cord.

The most difficult point in this case is to explain the localisation of the anæsthesia in the upper extremities. This loss of sensation affects both limbs symmetrically, involving two areas, viz. the outer side of the limb, supplied by the 5th cervical root, and the inner side, supplied by the 8th cervical and 1st dorsal, but omitting the intermediate portions which derive their sensory nerve supply from the 6th and 7th cervical roots. The explanation of this remarkable localisation, given by Dr. Ross, under whose care the patient was placed, and for whose inspiration upon both this and other subjects I cannot sufficiently express my indebtedness, is as follows: That the hæmorrhage did not extend far, if at all, below the level of the 5th root, the atrophic paralysis below this point being due rather to subsequent myelitis, which would produce less serious mischief, and would affect mainly the grey matter of the anterior horns. That thus we have, at or about the level of the 5th cervical root, a destroying lesion affecting nearly all the structures of the cord, but that below this point the conducting fibres largely escape. That the sensory fibres of the 5th nerve are injured immediately on their entry into the cord. That the fibres of the 6th and 7th roots, crossing obliquely as they do, and probably very superficially, are, at the level of the 5th roots, so near the periphery as to escape injury; but that the fibres of the 8th cervical and 1st dorsal nerves have probably already completely crossed over before reaching the level of the 5th root, and having thus acquired a deeper site have again come within the influence of the lesion. The great difficulty which this ingenious theory presents is that, on the left side of the body, there is no anæsthesia below the level of the 1st dorsal nerve-root, and that thus we have not sufficient evidence of compression or other injury of the sensory conducting paths on the right side of the cord. Possibly the explanation may be that the hæmorrhage or subsequent myelitis had taken a somewhat irregular direction, extending into the nuclei of some of the roots but not of others; or that, as in Case IV., there were two or more scattered hæmorrhagic foci.

We have only to note further, that here, as in Case VI., the cause of the intra-medullary hæmorrhage was apparently

an acute bend of the cord, due to the direct application of great violence.

CASE VIII.—*Traumatic Cervical Hæmatomyelia.—Temporary Paralysis of Limbs and Trunk.—Recovery.*

W. B., aged 40, was admitted to Mr. Heath's wards on Aug. 15th, 1885, suffering from the results of a fall estimated at a distance of 50 feet. He was a strong, healthy-looking man with no trace of previous illness. On the right side of the head was a deep scalp-wound, extending about 4 inches backwards from the forehead, and surrounded by much bruising. It may at once be said that under ordinary treatment this wound soon healed up. There was no unconsciousness, nor other trace of cerebral symptoms. All four limbs were, however, paralysed; the arms completely so; the legs could be moved in bed, but the patient could not stand. He had complete anæsthesia of the arms, abdomen and legs, but no loss of control over the rectum and bladder. The skin over the lumbar spine showed extensive bruising. Unfortunately, the notes of this case were very imperfectly taken, and there is no further information contained in them.

On the following day the temperature rose to 99·2° F., in the morning, and 100·8° F., in the evening: there was more power in the lower limbs and muscles of the neck. On Aug. 17th, he could move his legs quite easily and freely in bed, and there was no sign of paralysis of the neck, but both arms remained completely paralysed. Sensibility was improved, but there were tingling sensations in all the limbs. The temperature was lower. On the 18th sensibility appeared to have quite returned. The fingers and thumb could be moved fairly well on both sides, and there was slight movement of the whole upper limb. The temperature had returned to the normal, at which it afterwards remained. From this time there was a slow but steady gain in power, and on Aug. 28th, the upper extremities could be moved in any direction, but only feebly so. It was, however, some time before the legs were sufficiently strong to support him. He was first able to walk a little on Oct. 12th, and could then use his arms for such light work as holding a book, but they were still weak; there was no marked muscular wasting—merely the emaciation due to lying in bed; there was marked ankle-clonus, and exaggerated patellar, triceps and radial reflexes. The only affection of sensibility was some numbness of the inner sides of the calves, with tingling sensation in the fingers.

Three days later he was sent to the Convalescent Hospital at

Cheadle, where the constant current was applied to various parts of the limbs three times weekly, and where strength rapidly returned.

I again saw the patient on Jan. 16th, 1886, *i.e.* five months after his accident. He then looked very well and had returned to his work, but still felt weakly and had the sensation of tingling in the fingers; there was, however, no anæsthesia, and no difficulty in using the hand. He stated that at times he had some girdling sensation, which he referred to the region of the lower dorsal and upper lumbar nerves. The patellar and radial "deep reflexes" were exaggerated, but there was no ankle-clonus and no jerk on striking the triceps tendon.

The symptoms of this case are entirely explicable on the supposition, that the patient had a hæmorrhage into the cord, affecting the central grey matter at the level of the brachial plexus. This appears to have been small, and to have rendered itself obvious only by causing compression. There was no priapism, and no affection of the bladder or rectum, so that conduction to and from the lumbar centres was even less interfered with than in the two preceding cases. Anæsthesia also was very temporary, thus showing once more how rapidly the sensory fibres of the cord may accommodate themselves to slight pressure. It is interesting to note that as the anæsthesia passed off there was developed some "tingling" sensation, which we saw also in the last case under analogous circumstances. Here also, as in Case VII., we have some descending degeneration of the pyramidal tracts, due probably to pressure, and evidenced by the spastic symptoms in the lower limbs. That the effusion of blood was small, and did not extend to the anterior cornua, is indicated by the fact that there was no atrophy in the muscles of the upper limb. Unfortunately, the circumstances of the accident do not, in this case, allow of our exactly determining the nature of the injury to the spine, but it seems at least probable, from the presence of the severe scalp-wound, that the man fell upon his head, and that then there was an acute bend of the spine in its most vulnerable site, the whole weight of the body acting as a lever to bend the spinal column at this point. The bruise over the lumbar spine was clearly due to a less severe blow than that which caused the scalp-wound, and probably resulted from the body

striking the ground after the head. This is precisely the explanation given by Mr. Page of MM. Duménil and Petit's case of "concussion" already quoted; and, indeed, the last three of our cases appear to me to confirm most strongly the views set forth by Mr. Page in the article above referred to.

CASE IX.—*Traumatic Meningeal Hæmorrhage (?).—Paralysis of Limbs and Trunk.—Partial Recovery.*

M. H., a widow, aged 37, was admitted under the care of Mr. Jones, on May 10, 1886. Five weeks previously, she received two bullet-wounds from a revolver, probably fired at a distance of about two yards. One bullet pierced the left thumb and then passed through the lobe of the left ear, grazing the mastoid process and apparently at once escaping. The other entered immediately below and behind the left mastoid process, passing downwards and to the right. She was "immediately" paralysed in all four limbs, and when seen by a medical man was found to have complete loss of sensation below the neck, diaphragmatic breathing, and incontinence of urine and fæces. An attempt was made to find the bullet, but proved unsuccessful. The wounds healed well in about a fortnight, during which time the nervous symptoms were said to have remained unchanged. Calomel was occasionally administered to regulate her bowels, and her limbs were rubbed with oil.

On admission to the Infirmary five weeks after the injury, she was found to be a well-nourished and healthy-looking woman. In addition to the scars left by the above-mentioned wounds, she presented the following symptoms. At the back of the neck, some 3 inches to the right of the 5th cervical spinous process, was felt a hard nodule, apparently the bullet, lying quite superficially. She felt pain on moving the head, and tenderness of the cervical spine. There were no cerebral symptoms. The upper limbs were completely paralysed. The lower extremities were less absolutely so: on the left side she had some power of extension and flexion of the ankle-joint, and could raise the limb slightly from the bed; on the right side there was even less movement of the foot. The abdominal and intercostal muscles were also paralysed, breathing being diaphragmatic. There was no anæsthesia, but slight hyperæsthesia about the shoulders. The superficial and deep reflexes were normal. The limbs did not appear wasted. There was incontinence of urine and fæces. The temperature was normal, and remained so throughout the patient's stay in hospital. There were no circulatory or digestive disturbances.

A few days later, Mr. Jones removed the bullet without difficulty from the site above indicated. It proved to be a conical revolver-bullet, about ¾ in. long and flattened at the apex from contact with some hard substance. The wound healed readily, and gave no trouble.

On May 19th were noted involuntary twitchings of the legs, which were very constantly present and continued during her stay in hospital. On the 25th, she was found to have some slight power of movement of the fingers of her left hand, and increasing power in the movements of the feet. The faradic current was regularly applied to the limb muscles, the electric reactions of which were normal. Shortly after this she commenced to move the abdominal muscles, and to a slight extent the intercostals: the power of all of these slowly increasing. Towards the middle of June she became able to move slightly the fingers of the right hand, but there was little improvement elsewhere. She complained a good deal of occasional wandering sensations, of "pins and needles," and of the almost constant muscular tremors. From this time, until she went home on July 30th, there was no change, except a slight increase in the power of the already-acquired movements.

By kind permission of Dr. Martin, her medical adviser, I saw this patient again, at her own home, in September. As regards motor power, there was little or no change from the time of her leaving the Infirmary. The limbs were not more wasted than would be expected from her long confinement to bed, and the muscles everywhere presented normal electric reactions. The knee-jerk was much exaggerated, and there was ankle-clonus on both sides; the radial, ulnar, and triceps reactions were absent. She had complete control over her bladder and rectum. There was no anæsthesia. The pupils were normal. She complained greatly of pain in the various joints, but especially in the left elbow and ankle. The general health had remained good.

This case presents so many points of similarity to Case VII. that we are tempted to seek in the latter the explanation of the symptoms. There is the same sudden paralysis with anæsthesia, followed by spastic symptoms, the anæsthesia passing off gradually, the bladder and rectum, at first paralysed, recovering their functions. There is, however, this important difference, that, whereas in Case VII. we have evidence of destruction of a portion of the grey matter of the cord, causing atrophic paralysis and some permanent impairment of sensa-

tion, there are here no symptoms but such as might arise from compression of the cord. Further, in the present instance, the symptoms are absolutely symmetrical. We are thus led to look for some cause of general compression of the cord, at a point near the origin of the 4th or 5th cervical nerve. In Case VII., the compressing agent was a central hæmorrhage, causing destruction in its immediate vicinity. In the present instance we can hardly assume this to have been the lesion, as, if we do so, we must suppose an effusion of blood large enough to cause continued pressure on the whole of the structures of the cord, and yet so small as not to have extended into the anterior cornua. If, however, the hæmorrhage were meningeal, lying entirely outside the cord, it might very well cause such pressure without producing any destruction of its substance. Now, a moment's consideration will show, that the accident was of just such a nature as to produce this lesion. An examination of the neck, or of such a plate as that given on p. 302, vol. i., of Quain's 'Anatomy' (8th edition), will show, that if a bullet were to enter "immediately below and behind the left mastoid process," and pursue a straight course to a point "some 3 inches to the right of the 5th cervical spinous process," it would cross the vertebral column about the level of the 4th cervical spine, that is, about the level of the 5th cervical nerve, the upper limit of the paralysis and anæsthesia in our case. Now, the bullet was flattened at the apex as if it had struck some bone, and as it entered below the mastoid process, the only bone in its track would be a portion of the spine. It would then appear that the bullet struck the spinal column somewhere immediately above the 5th cervical nerve, and it probably did not penetrate the cord, or there would not have been the subsequent partial recovery. It appears to us, then, more than probable that it injured in its passage the spinal meninges, giving rise to hæmorrhage sufficiently profuse to compress the cord. It may be said that the instantaneous appearance of the symptoms negatives this view of their production, but we must remember that the only authority for this sudden onset is the patient herself, who was doubtless not at the time in a judicial frame of mind, having been shot by her husband, and having then to witness the latter commit suicide.

That she should fall is not surprising; she would then be confused, probably faint for some little time, and that time would be quite sufficient for the effusion of a quantity of blood from the rachidian veins. It is also possible that a small piece of bone may have been detached from one of the vertebræ and driven in upon the cord, so as to press upon without lacerating it which would naturally cause instant symptoms of compression, but which accords less satisfactorily than does the theory of meningeal hæmorrhage with the partial recovery which ensued.

In concluding a review of the above cases, there are a few points to which reference may be made, and for the consideration of which it is more convenient to regard the cases collectively.

We are at once struck by the relatively large number of cases of hæmorrhage within the vertebral canal, constituting at least three, and probably four, out of a total of nine cases of injury to the cervical region of the spine. The number of these cases occurring in actual practice is probably much under-estimated, many of them being regarded as instances of concussion of the spine, without definite organic lesion. The symptoms are frequently complicated, and without a careful consideration of the origin and course of the affected tracts, would appear to be inexplicable on the hypothesis of a single lesion. If, however, these symptoms be fully investigated with the assistance of an accurate knowledge of the anatomy of the cord, they will probably in most cases be found to be clearly due to some gross local lesion. It is to be regretted that of the numerous reported cases of spinal injury, a minority only are sufficiently detailed to permit of an accurate diagnosis, it being only too customary to use such loose expressions as "partial paralysis of the upper limbs."

Another cause of the comparative neglect of hæmatomyelia as a result of injuries to the spine, is the relative infrequency of post-mortem examinations verifying this condition. These cases are not nearly so fatal as fractures and dislocations, and their true pathology is therefore not fully cleared up. Thus we find that of our five cases of fracture all were fatal, whereas of the four cases of hæmorrhage only one died. Under these

circumstances the only possible means of diagnosis is the demonstration that all the symptoms may be due to a single focus of injury ; but where such demonstration is practicable, we submit that a diagnosis of hæmorrhage should always be preferred to one which regards the case as an instance of that vague and somewhat theoretical affection, "concussion of the spinal cord."

We notice also the tendency of hæmorrhage into the cord to occur not very far from the level of the 4th or 5th cervical nerve, and we think that a simple mechanical consideration will explain this localisation. In Case IX. the direction taken by the bullet was, of course, a pure accident, and we may therefore omit that case from consideration. But in all the other three instances (Cases VI., VII., and VIII.) of hæmorrhage, we have seen reason to believe that the cord lesion was the result of an acute bend of the cervical portion of the spinal column. Now, a glance at a properly-articulated skeleton or well-drawn picture of the spine will show that the most anterior of the cervical vertebræ are the 4th and 5th, with their intervening disc. If, now, this region of the spine be over-extended, either from a blow on the back of the neck, or from forcing back of the head or trunk, these two vertebræ will form the apex of the angle which is produced, and here, therefore, will be the most acute bend of the cord. Thus it happens that in all such injuries the cord is more stretched at this point than at any other, and is therefore more subject to a laceration of its blood-vessels, and the production of a hæmorrhage.

Finally we may note the relative frequency in our cases of certain well-recognised symptoms of injury to the spine.

As regards *external deformity*, we find this present only in one case (Case I.), there being in none of the others the slightest local indication of the severe injury which had been sustained.

Hyperæsthesia was observed in one case only (Case IX.), but in all the three cases which recovered (Cases VII., VIII., and IX.), there was observed a sensation of *tingling* in the parts previously anæsthetic, as the anæsthesia was gradually passing away.

Priapism was present in all the cases of fracture during the remainder of life. Of the three cases of hæmorrhage occurring in males, it was present in one alone (Case VI.), and even here it existed for about twenty-four hours only. It would thus appear that the fibres connecting the lumbar centre with the brain are situated at some considerable distance from the centre of the cord, so as either altogether to escape, or readily to recover from the pressure of a central hæmorrhage.

In the same way we find that the fibres connected with the bladder-centre are not liable to suffer from prolonged compression by a central hæmorrhage. Thus there was permanent *retention of urine* in all the cases of fracture. On the other hand, looking at the cases of hæmorrhage, we find retention lasting one day only in Case VI., and four days in Case VII.; while in Case VIII. there was no bladder trouble at all. In Case IX., a female, there was incontinence for some weeks, but not permanently. Can it be that the implicated fibres are situated near the periphery of the cord, that they thus escape compression or rapidly recover in the cases of central hæmorrhage; but that, owing to the hæmorrhage in Case IX. being meningeal, they are more directly exposed to a pressure, and therefore incapacitated for a longer period?

The present cases throw no light on the chemical changes which the urine may undergo. Two of them were examined at an early period for sugar and albumin, with negative results. In Case I. decomposition and alkalinity set in about the end of the first week, but the other cases give no indication of the period at which this change usually occurs, all having either died or regained control over the bladder before the fourth day, with the exception of one which was only seen long after the injury.

Bed-sores were noted in Case I. at the time of admission, four days after the accident; all the other cases of fracture died within a few hours, before their development was possible, and they did not appear in any of the cases of hæmorrhage.

The condition of the *pulse* was noted in Cases I., III., V., and VII. In the first three of these it had the characters usually described in lesions of the cervical cord, being slow, full and of low tension. In Case VII. it was quick, but the

observations were only made after the setting in of the febrile reaction due probably to myelitis. In Case IX. it was quite normal at the time the patient came under observation, five weeks after the accident. Case III. is, however, evidence that the pulse-rate may increase with rise of temperature, a pulse of 130 being rapid even with a temperature of 103°.

As is usually the case in spinal injuries, the *temperatures* observed were sufficiently remarkable, appearing to bear no relation to the nature and site of the injury; it is unnecessary to repeat the figures given above, no classification of cases being as yet possible.

The condition of the *pupils and palpebral fissures* is interesting, not only from a scientific point of view, but also because changes are very constant and are highly unlikely to be simulated in any case of attempted deception. Whether or not we accept the existence of a cilio-spinal centre in the cord, there can be no doubt that dilator fibres for the iris, and fibres supplying the muscle of Müller pass downwards to about the level of the last cervical or first dorsal nerve-root, and thence outwards to join the sympathetic by means of the *rami communicantes*, and that these fibres are liable to be injured by any lesion of the cord above this level. The affection may, however, be overlooked if bilateral, being distinctly visible only in a dim light, because in bright light contraction is normally present and therefore would not be remarkable. In Case I. we find imperfect dilatation of the pupil on both sides, lasting until the closing stages, when there was dilatation, owing probably to the iris assuming the position of rest from paralysis of all its muscles, preceding death. In Case II. the pupils were said to be "normal," but this was probably owing to their not being properly examined in diffused light. In Cases III., V., and VI. there was no doubt about the contraction both of the pupils and of the palpebral fissures. In Case IV. the pupils were dilated, but death occurred before the subsidence of the first shock of the accident. In Case VI. the contraction was much more marked on the right side, coincidently with the greater extension of the hæmorrhagic focus on that side. In Case VII., where the lesion was more extensive on the left side, the left pupil only was contracted.

In Cases VIII. and IX., where the injuries were less severe the pupils had altogether escaped. In Case III. the paralysis of Müller's muscle gave rise to a very remarkable sensation of softness and compressibility of the eyeball.

The last symptom to which I wish to refer is the condition of the cutaneous reflexes and tendon reactions below the seat of the lesion. On this subject we have notes in six of the cases only. We see at once that there is an important distinction to be drawn between the condition of the reflexes in the earlier and in the later periods after the injury. As regards *cutaneous reflexes*, they were noted as absent immediately or very shortly after the accident in Cases I., V., and VI., and they were lost later on in Cases VII. and IX.; beyond this there was no examination, but the results are exactly in accordance with the common observation that cutaneous reflexes are usually diminished or lost in cases where communication is cut off between the cerebral cortex and the periphery. The reason of this change is perhaps not yet satisfactorily explained, but as it is generally recognized as a matter of daily observation in all diseases of the nervous system, we need not dwell upon it longer at present.

As regards the *tendon reactions*, we find a most interesting condition. In Cases I., V., and VI. they were absent immediately after the occurrence of the injury, their early condition not being noted in any of the other cases. On *à priori* grounds we would expect that a complete transverse lesion of the cord, high up, would, by removing the influence of cerebral inhibition, tend to increase the activity of the tendon reactions—the knee-jerk, and ankle-clonus. Such is the change soon after a transverse myelitis, after compression of the cord, and after degeneration of the lateral columns. Here, however, the exactly opposite condition is produced. Probably the true explanation of this peculiarity is, that the shock of the accident acts as a powerful depressant of reflex action, and is thus more than able to counterbalance the increased activity which the reflex centres should normally acquire on being relieved from the repressing influence of cerebral inhibition. The facts would thus appear to be of the same order as the well-known observations of Goltz, that upon section of the

spinal cord of the dog in the neck the lumbar reflex centres for the bladder, rectum, and generative organs are temporarily paralysed. This condition of paralysis of reflex action from shock is constantly seen clinically in cases of retention of urine following upon operations or injuries of quite distant parts—as, for instance, in a case which I have seen quite recently, where retention of urine lasted for two days after the performance of Mac Ewen's operation for genu valgum. If such be the true explanation of this loss of the deep reflexes, we should expect them soon to be restored to their normal condition, but from the very nature of the cases this is a change which can seldom be observed. Thus Cases V. and VI. died within a few hours of the accident, there being no opportunity for the reflex centres of the cord to recover their functions. Case I., it is true, lingered longer, but he remained throughout in a condition of depressed vitality not favourable to energetic reflex action, and we know well that in many cases of general depression the reflexes are diminished or absent. Further, it is to be remembered that the determination of a slight knee-jerk is by no means easy in a patient necessarily recumbent on a water-bed. But, turning to some of the later cases, we find evidence that the knee-jerk does, as a rule, return before very long, the best example being Case IX., where at the fifth week after the accident the knee-jerk was apparently normal in degree. Finally, we find that in older cases, where there has been time for the development of a descending degeneration of the pyramidal tracts, there is, as is usual under such circumstances, a well-marked intensification of the tendon reactions, as evidenced by Cases VII., VIII., and IX.

SEQUEL TO A "CASE OF OPHTHALMOPLEGIA, COMPLICATED WITH VARIOUS OTHER AFFECTIONS OF THE NERVOUS SYSTEM."[1]

BY J. S. BRISTOWE, M.D., F.R.S.

In my paper I gave reasons for believing the symptoms in this remarkable case to be due to functional disease of the nervous system; and remarked, "A practical advantage in regarding the cases I have cited as functional is, that it fortifies me in the hope, so long as the survivor" (Gertrude H.) "lives, that she may yet recover."

On reference to my paper, it will be seen that the patient had undergone no improvement when she passed from under my care on the 2nd of February, 1885. At my request, she was then admitted into St. Bartholomew's Hospital, where she remained for several weeks without material, if any, change. I had authentic information of her condition not long after she left that institution, at which time also no improvement had taken place. Shortly afterwards, the family removed from the East of London to Walthamstow, and I lost sight of her. But latterly rumours have reached me, through some of the hospital nurses, between whom and her family there has been kept up an irregular correspondence, that the girl had recovered; and on the 16th of November she came to St. Thomas's with her mother to pay me a visit.

She was thinner than she had been, and pale, but otherwise looked quite well. There was no paralysis of the eyes, which worked freely in all directions and consensually; the pupils were equal, and acted to light and accommodation. I thought the tongue, when protruded, tended slightly to the right, but the deviation was scarcely perceptible. She used both arms

[1] Case of GERTRUDE H. 'BRAIN,' October 1885, p. 328.

and both hands with equal facility; and said that her right hand was as useful as her left, but its grip was certainly not so powerful as that of its fellow. She walked perfectly well; but the knee-jerks were perhaps over-brisk. There was no anæsthesia. She had quite lost her headache; and never now complained of sickness or giddiness. It appears that she had had only two fits after leaving St. Bartholomew's; the first, six weeks after returning home, the second, about two months subsequently. I could not ascertain the order of events in her progress towards recovery; but her recovery seems to have been gradual; and she has been well and able to work like any other healthy young woman for the last twelve months.

Clinical Cases.

A CASE OF HYSTERO-EPILEPTOID ATTACKS, WITH CONVULSIONS PRODUCED BY LOCAL IRRITATION OF THE SKIN.

BY G. PARKER, M.D.,

Late Resident Medical Officer to the Children's Hospital, Paddington.

In the following case[1] there were two points of interest. 1. A convulsive seizure could be brought on by irritating a spot on the chest, but only if a sufficient time had elapsed since the last attack. If the epileptogenous zone were touched within about an hour from the previous seizure, no effect took place; but after an interval sufficient to permit a certain accumulation of energy, as it were, the patient was at once struck down if touched on a spot between the right nipple and axilla. 2. Very frequent seizures of a hystero-epileptic character occurring in a child, whose family history pointed rather to true epilepsy than to a syphilitic taint, yielded at once to mercury and potassium iodide, after numerous other remedies had been tried to relieve them without success.

Amy R., a healthy-looking child of 11, presenting no signs of premature development, was admitted as an out-patient on March 8. The mother stated that she had of late been subject to convulsive seizures, falling down with a scream at all hours of the day and night. The family history showed that the father's father had been subject to fits, during one of which he was drowned by falling into a river. The father denied any specific taint. Patient is the fourth in a family of nine children. The eldest is now 17, six are alive and healthy, none have had any fits. The mother had no miscarriages. Patient was always quite healthy till three years ago, when early one morning she became suddenly insensible and convulsed. Similar attacks occurred at intervals during six months. No exciting cause was known, but the mother says she noticed that two upper canine teeth were cut at this time.

[1] This case was under the care of Dr. Laycock, by whose kindness I am allowed to publish the observation.

One month ago, without any discoverable cause, the fits began again. On an average one every hour has occurred during the night, nine during the day. These numbers have remained approximately the same. She generally falls down with a scream, if standing or sitting, and has cut herself in doing so, but has never bitten her tongue or passed her water during a fit. She lies convulsed for one or two minutes; the left arm is flexed and drawn across the chest, the hand grasping forcibly the right side; the trunk is bent forwards to the right, and the head brought down on the chest; the right leg is kicked to and fro spasmodically, especially in those fits which occur during sleep. The movements, in fact, resemble in an exaggerated form those which would be made by a child when tickled in the right axilla and trying to save itself. The mother has noticed that if she is touched at a spot on the thorax between the right nipple and the axilla, an immediate attack comes on, provided that none have occurred within an hour or two previously. This was tested in the consulting room, and a fit induced. She is said to have frequently retained consciousness through her fits.

March 25.—Admitted as in-patient, not having improved under treatment. *Present condition.*—She appears to be well-nourished and cheerful; the general intelligence and memory are very good; bowels regular, appetite fair; nothing abnormal noted in the respiratory or circulatory systems; no signs of approaching catamenia; says that she has sometimes just before a fit a creeping sensation passing up towards the right axilla.

March 26.—She had five fits while in the hospital during the afternoon of yesterday, and six more in the night, each one lasting some seconds only. They commence with a groan, the head falls forward, the left arm is flexed across the chest and the hand grasps the right flank, the mouth and lower part of face are drawn into a sort of grin. To-day a fit was induced by touching the chest in the region of the right nipple. The conjunctivæ were quite insensible during some of the attacks. Seventeen fits occurred in the 24 hours, each lasting about 60 or 90 seconds. She is being carefully watched to discover the existence of masturbation, or any allied cause for the attacks.

March 27.—Seven fits during the day. She is apparently quite insensible to pain during the attacks if tested by a pin. There is no cyanosis or stoppage of respiration.

March 31.—There is tenderness in the region of the left ovary. The left pupil is distinctly larger than the right, she has apparently red blindness in left eye, and the hearing is

only half as acute with the left ear as with the right. There is no albumen in the water.

April 2.—Pot. bromide up to gr. xx. in the dose; am. and sodium bromide, belladonna, ergot, oil and steel wine, and valerian are being tried in succession. A slight improvement has followed the use of two or three of these remedies, but they all fail to act after the first few days. The fits to-day are fewer, but stronger, and followed by much exhaustion, which has not been the case previously.

April 5.—One fit last night continued for about five minutes, and was accompanied by foaming at the mouth. The numbers of the attacks from the first inst. have been for each 24 hours 16, 19, 20, 21, 19. The bowels are rather confined, and the temperature is nearly a degree above normal.

April 15.—The daily chart records from 10 to 20 fits every 24 hours. There is full power over the sphincters, knee-jerks taken after a fit were normal; no ankle-clonus is present. No impaired intelligence or aphasia, and no loss of sensation have been made out. Between the attacks she eats and plays like a child in perfect health. When one comes on, she falls suddenly forward with a scream, remains rigid and huddled together for about a minute, has generally some convulsive movements of the arms and legs, chiefly on the right side; the left arm is often dragged over as before. The right side of the face twitches and the eyes are turned to the right. Afterwards she appears to wake up with a smile, and smooths her clothes mechanically, or if it is night, she turns over and falls asleep again quietly. On examination by the ophthalmoscope the discs were seen to be normal, the choroid was thinned at the periphery, and a few spots of pigment appeared through it. R. $\frac{6}{12}$ m.; 5 D. $\frac{6}{6}$ pi. Left, $\frac{6}{18}$ n.; 1 D. $\frac{6}{6}$ pi.

May 4.—The fits are much fewer in the day-time; at night she groans and mutters during the short time they last. The limbs become rigid at the commencement of an attack, and are then flexed. At times there is much exhaustion. In other respects there is no change. Nitrite of sodium is now being tried.

May 13.—Faintness and cyanosis having been produced, the nitrite was stopped finally to-day. The attacks are again more frequent yesterday and to-day.

May 15.—To-day 25 fits are registered in the 24 hours.

May 16.—To-day 8 fits only.

May 24.—The faradic current was applied sharply to spine and limbs.

June 1.—Frequent short applications of the battery, both by night and day, after fits have been tried. The fits, which have

for some time been getting fewer in the day-time, now cease to occur except at night, but the number in the 24 hours remains the same. Urethan, valerian, the bromides, and camphor monobromide have all been tried without success.

June 14.—From 6 to 28 attacks occur nightly. To-day the liquor hyd. perchlor. ʒss, potassium iodide gr. iii, and decoction of cinchona were given for the first time.

June 18.—The attacks have fallen to one in the 24 hours.

June 19.—No fits at all to-day.

June 30.—No more attacks; patient a little weak, but otherwise quite well; ordered to become an out-patient, and to continue her medicine.

July 20.—Remains perfectly well. Mother states that she has cut four molar teeth this year.

Sept. 6.—Has had no recurrence of the fits, and remains in perfect health.

The disappearance of the attacks when treated with mercury may of course have been only a coincidence, but the suddenness of the change rather points to a causal relation, possibly by the reduction of some enlarged gland. The induction of the fits by touching a special area resembles the effects produced in certain guinea-pigs under Dr. B.-Séquard. In them, after the division of the sciatic or other large nerve epileptic seizures occurred when the skin of the cheek was irritated; and similar cases have occurred in human beings.[1] It was, however, obviously undesirable to encourage these tendencies in the patient, but sufficient trials were made to establish the fact clearly, as well as the existence of a period in which irritation had no effect. Besides the problems suggested by this period of stable equilibrium, the child's statement, that "a creeping feeling" occurred before some of the fits in the region where a touch would often bring them on, is important with regard to the nature of an aura. Either what is subjectively known as an aura is purely central in origin, and if so, we must say that there was no analogy between it and the touches in this case, though both were equally followed by fits; or it is some local impression, such as the friction of the clothes, which, occurring when the central irritation is at its height, is perceived abnormally, and is the determining cause of a fit, just as much as the touches were.

[1] See, too, a paper by Dr. Arthur Gamgee in the 'Brit. Med. Journal,' vol. ii. 1878, on Hystero-Epilepsy, and the recent experiments by Mr. V. Horsley discussed before the Pathological Society, Nov. 1886. Is there not an analogy between the state of the central nervous system thus produced, and the tremor which appears peripherally for some time after section of a trunk nerve?

A CASE ILLUSTRATING THE DIFFERENTIAL DIAGNOSIS OF TUMOUR OF THE CORD AND TUMOUR OF THE CAUDA EQUINA.

BY JAMES OLIVER, M.B., F.R.S. (EDIN.).

THE cord itself, as such, ceases to exist beyond the level of the lower part of the body of the first lumbar vertebra; whilst the cauda equina forming its continuation, is made up of the roots of the lumbar and sacral nerves as they descend to their respective foramina of exit. The symptoms complained of, and physical signs manifested by, a patient, the subject of a neoplasm invading either the spinal cord or cauda equina, necessarily vary according to the level at which the nerve-tract becomes compressed and so disturbed. The existence, however, of a difference in the structural arrangement of the nerve-fibres in the cord proper and its continuation, the cauda equina, produces a marked variation in the evolution and manifestation of symptoms, resulting from the presence of a new growth invading these two totally distinct parts of the spinal tract.

The bundles of nerve-fibres forming the cauda equina are like those composing any large peripheral nerve-trunk, both motor and sensory in character, which, continuing throughout their course perfectly distinct, end in their respective peripheral expansions, motor or sensorial, free anastomosis apparently of the corresponding terminal filaments being effected.

In the cord proper, however, the motor and sensory tracts do not long continue in apposition, but follow a different course in this structure; decussation, more or less complete, in the case of both, occurring, proximally with the motor, and distally with the sensory, in respect to the higher centre. The motor tract coming from one or other hemisphere of the brain, decussates with its fellow of the opposite side somewhere in the medulla oblongata, and is afterwards transmitted without any further appreciable decussation down one half of the cord, to supply the muscles of that same side of the body. The sensory bundles, on the other hand, resulting from a union of fibres coming from peripheral expansions, enter the cord at

varying levels, according to the part of the body in which they take origin, pass almost immediately to the opposite half of the spinal cord, and are thence transmitted to the brain, without further recrossing, in that part of the cord away from the side of the body in which they originate. The motor tracts, as they descend in the cord, give off nerve-fibres to the same side of the body, whilst the sensory, as they ascend, accumulate from the opposite half of the body. If, therefore, one half of the spinal cord be divided completely, the motor fibres supplying that same side of the body below this level are severed from the higher centre, whilst the sensory fibres corresponding with sensorial expansions of the opposite side of the body are interrupted in their course. When a patient suffers from slow compression of the cord—the result of invasion by a neoplasm—the clinical facts occasionally illustrate beautifully this physiological dogma, as in a case which recently came under my care, the leading points of which I shall here relate.

Henry I. ——, æt. 40, two years ago began to complain of pain in the back, extending round the left side, and following apparently the course of the eleventh and twelfth dorsal nerves. Soon afterwards he experienced sensory disturbances—pins and needles—in the right foot. This peripheral sensation gradually extended up the leg, and in the course of a few months he remarked that there was total loss of feeling in the right leg throughout. He was still able to walk about well. Gradually, however, he noticed a tendency to drag the left foot, and in the course of six months he had lost completely all power over the left leg. As yet he had experienced no motor impairment of the right lower limb, nor sensory disturbance in the left. One month later, he noticed a tendency to drag the right, and simultaneously, that he had lost sensation in the left; he had never, at any time, however, experienced any abnormal sensations in the left leg, as in the case of the right. The loss of volitional power over the right leg gradually progressed, and the patient now, two years after the initial manifestation of symptoms, is completely paraplegic, with a total loss of sensation in both legs.

The urine has to be drawn off.

No wasting of muscles of the limbs, which react sluggishly to the faradic and constant currents.

Skin plantar-reflex increased.

Knee-jerk well marked—somewhat augmented.

Ankle-clonus can be developed, although feebly.

In the case of tumour invading the cauda equina pain may be occasionally, although not invariably, complained of. There is noted gradual loss of power in the lower limbs, beginning at first in one leg, and eventually extending to the other.

Loss of volitional power is accompanied by a gradual, or sometimes rapid wasting of muscle tissue. These muscles, over which the patient has little or no volitional control, fail to respond to the faradic or constant current, whilst those which continue to respond to the volition of the patient, although somewhat enfeebled, will still react to electric excitation, faradic or galvanic. Usually there is some defect in the tactile, and painful sensibility; as, however, sensation may apparently be carried on by a free nerve-anastomosis, this may not be a marked symptom of the disorder. When there is evidence of sensory disturbance, it is experienced first, and, as a rule, much more markedly in the limb which first shows signs of motor enfeeblement; and not, as in the case of tumour invading the cord, primarily in the limb opposite to that which reveals initially the signs of motor impairment.

The bladder in such cases, as in the case of tumour invading the lumbar enlargement of the cord, has its functions disturbed, the patient being as a rule unable to empty this viscus of its contents.

There is absence of skin plantar-reflex, no ankle-clonus, and invariably it is impossible to elicit any knee-jerk.

Abstracts of British and Foreign Journals.

Philosophische Studien. Herausgegeben von Wilhelm Wundt. Dritter Band; pp. 691. Leipzig: Wilhelm Engelmann, 1886.—Prof. Wundt's 'Archiv' contains much that is of interest to the readers of 'Brain,' and must not be ignored on the supposition that it is given over to metaphysical discussion and commentaries on Kant. The editor's name is sufficient indication of the scientific point of view adopted, and he expressly states at the close of the first volume that the chief aim of the 'Studien' is the building up of experimental psychology. In looking over the three volumes so far issued[1] it will be seen that about half of the papers are accounts of experimental work done by Professor Wundt's pupils in the psychological laboratory at Leipzig, while about half of the remainder discuss methods of experimental research. Valuable results in psycho-physics had been obtained by individual workers, or published from physiological and physical laboratories. But Professor Wundt took a step of great importance in founding in 1879 a special laboratory for psycho-physical research. A similar laboratory was started in 1883 at Johns Hopkins University, and others are about to be formed at Cambridge and the University of Pennsylvania; but the most systematic and valuable work done during the past seven years in psycho-physics comes from Professor Wundt's laboratory, and is described in the 'Philosophische Studien.' It would lead too far to give even a brief summary of this work, but it may be worth the while to indicate its scope so that those interested can refer to the original papers. About half of the experiments described are directed towards determining the time taken up by mental operations, a direction of research to which the term 'psychometry' might be confined. The reaction time has been measured under varying conditions, as also the time

[1] Numbers are published as material is accumulated; during the past five years twelve such numbers have been issued, making up the three volumes.

needed in perceiving, willing and in the association of ideas. On the work with which the names of Weber and Fechner are associated, that is the relation between the strength of the stimulus and the intensity of the sensation (psycho-physics in its narrower sense), experiments on light and sound have been published and others are now being carried on. The analysis of sensation, to which we may reckon contrast, has proved itself to be one of the most fertile fields of investigation, including as it does the researches of Helmholtz on sound, and the many valuable experiments on the retina and sense of sight. The extent and limits of consciousness is a subject of considerable interest and open to experimental study. Investigations have been made at Leipzig, and are still being prosecuted on the time-sense, under which we may include the estimation and comparison of intervals and the time relations in which sensations stand. Lastly, the conditions of memory, on which Ebbinghaus has made interesting experiments, may be mentioned. These six branches of research are those which have been taken up at Leipzig, and do not of necessity cover the whole field of psycho-physics, which is itself but one branch of psychology.

Experimental psychology enjoys the advantage of having its way to some extent cleared by physics and biology, and can borrow from these sciences apparatus and methods; but any one working in psycho-physics will find that he must himself devise and control the greater part of the apparatus needed, not finding ready, for example, such a purely physical and elementary fact as the time it takes an electro-magnet to attract its armature. Still more must the experimenter rely on himself in arranging methods; these depend to a large extent on the very consciousness we wish to investigate, and their advantages and shortcomings can often only be seen after the experiments have been made. I mention these facts, to explain why the work described in the 'Studien' is not all of equal value; when similar experiments are continued by the same or different workers, improvements in method and apparatus are constantly being made, so that the later results are the more accurate and valuable.

I give below a classified list of the papers which describe experimental work. In some cases a paper covers more ground than the heading under which it is entered.

J. McKeen Cattell, Ph.D.

PSYCHOMETRY.

'Ueber die Appercceptionsdauer bei einfachen und zusammengesetzten Vorstellungen.' Max Friedrich. i. 39–78.

'Experimentelle Untersuchungen über die Association der Vorstellungen.' Martin Trauts-holdt. i. 213–251.

'Ueber die Einwirkung einiger medicamentöser Stoffe auf die Dauer einfacher psychischer Vorgänge.' Emil Kraepelin. i. 417–463 and 573–606.

'Ueber die Unterscheidung von Schallstärken.' Ernst Tischer. i. 495–513.

'Ueber die einfache Reactionszeit einer Geruchsempfindung.' W. Moldenhauer. i. 606–615.

'Die zeitlichen Verhältnisse der Willensthätigkeit.' Julius Merkel. ii. 73–128.

'Ueber die Zeit der Erkennung und Benennung von Schriftzeichen, Bildern und Farben.' James McKeen Cattell. ii. 635–651.

'Ueber den Einfluss der Reizstärke auf die Dauer einfacher psychischer Vorgänge mit besonderer Rücksicht auf Lichtreize.' G. O. Berger. iii. 38–94.

'Psychometrische Untersuchungen.' James McKeen Cattell. iii. 264–305 and 452–493.

PSYCHO-PHYSICS.

'Zur Frage der Gültigkeit des Weber'schen Gesetzes bei Lichtempfindungen.' Emil Kraepelin. ii. 316–327.

'Die Methode der richtigen und falschen Fälle in ihrer Anwendungen auf Schallempfindungen.' Gustav Lorenz. ii. 394–475.

ANALYSIS OF SENSATIONS.

'Untersuchungen über den Farbencontrast vermittelst rotirender Scheiben.' Bruno Schmerler. i. 279–305.

'Bemerkungen über die Messung von Schallstärken, mit Rücksicht auf psychophysische Versuche.' Ernst Tischer. i. 513–556.

'Die Messung von Schallstärken.' Paul Starke. iii. 264–305.

'Ueber die Anwendung der Methode der mittleren Abstufungen auf den Lichtsinn.' Alfr. Lehmann. iii. 497–534.

LIMITS OF CONSCIOUSNESS.

'Untersuchungen über den Umfang des Bewusstseins bei regelmässig auf einander folgenden Schalleindrücken.' Georg Dietze. ii. 362–394.

'Ueber die Trägheit der Netzhaut und des Sehcentrums.' James McKeen Cattell. iii. 94–128.

TIME-SENSE.

'Untersuchungen über den Zeitsinn.' Julius Kollert. i. 78–90.

'Neue Versuche über den Zeitsinn.' Volkmar Estel. ii. 37–66.

'Zur Lehre vom Zeitsinn.' Max Mehner. ii. 546–603.

'Ueber die Zeitverhältnisse der Apperception einfacher und zusammengesetzter Vorstellungen, untersucht mit Hülfe der Complicationsmethode.' Woldemar von Tchisch. ii. 603–635.

MEMORY.

'Untersuchungen über das Tongedachniss.' H. K. Wolfe. iii. 534–572.

Abstracts of Papers on Nervous Diseases published in St. Thomas's Hospital Reports from 1875 to 1884 (vols. vi. to xiv. inclusive). By W. B. Hadden, M.D. (Lond.)

1875.

"Some Cases of Sudden Recovery from Mental Disease." By W. Rhys Williams, M.D.

The recovery in each case was permanent, and in this respect differed from many of the recorded cases in which recovery was transient.

1876.

"Notes on Cases of Nervous Disorder." By W. M. Ord, M.B, F.R.C.P.

The author first discusses reflex irritation of the skin, and the part played by reflex irritation in producing certain morbid conditions of the skin. Four cases are cited in which eczema appeared to arise from irritation, mechanical and catarrhal, of the urinary organs and passages. Acne results from irritation of the mucous membrane of the genital passages, and the psoriasis and prurigo which are common at the climacteric period, are probably related with local changes. There are reflex relations between eczema and bronchial catarrh, these conditions sometimes alternating in the same individual. Patchy baldness of the scalp is often associated with anal pruritus. The reflex influence exerted by the skin on internal organs is illustrated by several cases in the author's practice. He concludes that it is possible to rouse "reflex trophic influence in wasting limbs by stimulation of a dormant central ganglion through the peripheric nerves."

"Intra-Cranial Aneurysms." By Thomas B. Peacock, M.D., F.R.C.P.

This paper contains an admirable *résumé* of this subject up to date. No less than eighty-six cases are summarised, and the conclusions to be drawn from them are given in great detail. The paper is well worth consulting for reference.

1877.

"Statistical Report of Cases of Chorea." By Thomas B. Peacock, M.D., F.R.C.P.

This is an elaborate analysis of ninety-two cases of chorea occurring in the author's practice. It is well adapted for reference, and would prove an admirable guide for future observers.

"On Simple Meningitis." By W. S. GREENFIELD, M.D. Lond.

Two groups of cases are embraced in this paper: (1) those that are truly primary; (2) those arising as a complication or sequela of acute disease, or as the result of some blood contamination. In the first class are included sporadic cases of cerebro-spinal meningitis, and the form which occurs in infancy without known cause; in the second class, which is mainly discussed and illustrated, are grouped the cases depending on such conditions as pneumonia, rheumatism, pyæmia, septicæmia, syphilis and Bright's disease. The paper contains some excellent and suggestive observations on the pathology of this affection.

1878.

"Cases of Chorea." By J. S. BRISTOWE, M.D., F.R.C.P.

The author remarks that he has never seen a post-mortem examination in chorea without heart disease having been detected, although its existence is not always recognised during life. In one of the cases given there was complete loss of speech, and the choreic movements were most marked on the right side. Attention is drawn to cases in which paralysis (paraplegia or hemiplegia) may precede or replace the chorea. Impairment of common sensation, general or unilateral, is sometimes observed, resembling closely that found in hysteria. Absolute insanity supervenes in rare instances. In two of the cases recorded, epileptic attacks existed, and in one of these insanity came on. In the other, the choreic symptoms were preceded, and in part, accompanied by a succession of epileptic fits. Occasionally chorea becomes chronic, and in two such cases seen by the author the lower limbs became paralysed and stiff, suggesting degenerative change in the lateral columns of the cord.

1879.

"On Hysteria and Hystero-Epilepsy." By WILLIAM H. STONE, M.B., F.R.C.P. "Also Further Remarks on Hystero-Epilepsy."

The author discusses the term "hysteria" at some length, showing how its meaning has become modified, owing "to the tendency to metaphor and metathesis which is the besetting sin of medical terminology." A classification of functional neuroses is suggested, each group being illustrated by cases. The author narrates his experience of *le transfert* as seen in Charcot's wards at Salpêtrière, and then proceeds to point out what he considers

weak points in the demonstration. In the second paper the author gives two typical cases presenting the symptoms described by Charcot, which came under his notice since the first paper was written. In one of these cases the anæsthesia disappeared when a magnet was placed close to the affected limb. Elaborate precautions were taken to exclude the effects of expectant attention.

1881.

"On the Use of the Continued Current in Diabetes." By William H. Stowe, F.R.C.P., and Walter J. Kilner, M.B.

In the two cases recorded an ascending current from the nape of the neck to the forehead was employed, the negative pole being placed in the former region, the positive in the latter. Some temporary improvement seemed to result from this treatment; but in the first case a large carbuncle formed at the back of the neck where the negative pole had been applied, and the patient died.

"Contribution to the Pathology of Double Optic Neuritis." By Walter Edmunds, M.D.

This paper is based on a case of head injury which proved fatal in twenty-four hours. There was a fracture of the middle fossa of the base of the skull, but it did not pass into the orbit or into the immediate neighbourhood of the optic nerves. These latter exhibited well-marked early inflammatory changes. There was some basal meningitis, and the author believes that the simplest explanation of the appearance described "is that the inflammation spread from the inflamed meninges to the tissue between the nerve sheaths."

"Nerve-stretching in a Case of Spinal Meningitis with Ataxic Symptoms, due to Injury." By W. H. Armstrong.

The author claims that this was one of the first cases of nerve-stretching done in this country, and the result was more successful, than that of most cases hitherto published. By the operation the patient recovered cutaneous sensibility, the lancinating pains were much relieved, and the power of locomotion was greatly improved. In this case the left sciatic nerve was stretched, and the author is inclined to believe that an effect is produced on the nerve centre, not on the periphery.

"On Cases of Injury to the Optic Nerve." By Edward Nettleship.

The author points out that the optic nerve seldom undergoes repair and regains its function after injury. This depends in

great measure on the usually severe nature of the injury, possibly also on the anatomical peculiarities of the optic nerve. When the nerve is divided in front of the optic foramen, the movements of the eyeball may interfere with the apposition of the cut ends. The paper is illustrated by a series of valuable cases.

1882.

"On Paraplegic Rigidity in Hemiplegia."
By W. B. Hadden, M.D.

The author calls attention to the fact, that a lesion in one cerebral hemisphere gives rise to direct as well as crossed paralysis; and that in late hemiplegic rigidity the lower limb of the opposite side may become the seat of ankle-clonus, and occasionally even becomes contractured like its fellow. It is also observed that in bilaterally associated nerve-nuclei, irritation on one side, as in the case of inhibition, reacts on the opposite side. Moreover, bilaterally associated nerve cells are less apt to undergo atrophic changes than cells not bilaterally associated. It seems likely that paraplegic rigidity in hemiplegia depends on the variations in the decussation of the pyramidal-tract fibres in the medulla, and not on a secondary decussation of the motor fibres in the spinal cord. It is possible that the fibres of the direct pyramidal tract enter into relation with the multipolar cells in the lower part of the cord.

"Some Effects of Brain Disturbance on the Hand-writing."
By W. H. Stowe, F.R.C.P.

This paper contains some interesting observations on agraphia, illustrated by the author's personal experience during an obscure illness.

"Clinical Remarks on so-called Painful Paraplegia."
By J. S. Bristowe, M.D., F.R.S.

The paraplegia to which attention is called depended in all the five recorded cases on malignant growths, involving the spinal cord or the nerves proceeding from it. In two instances the lumbar vertebræ were affected, and in the remaining three the growth originated in the periosteum of the ilium.

"Paralysis of Serratus Magnus." By H. H. Clutton.

Among the causes of the paralysis the author gives:—typhoid fever, progressive muscular atrophy, blows and strains, and possibly infantile paralysis. In three cases observed by him no definite cause could be assigned. The anatomical and clinical aspects of

the paralysis are fully discussed, and the views of Erb, Duchenne, Bäumler, Vivian Poore, and others, compared and criticised.

"On the Electrical Resistance of the Human Body." By W. H. Stowe, F.R.C.P.

This is an abstract of a paper read before the British Association at Southport. The author believes that the electrical resistance of the human body is enormously overstated. The obstacles to its accurate determination and the means adopted to overcome them are stated in detail. The paper will be of interest to experts.

1883.

"Two Cases of General Atrophy treated by the 'Weir Mitchell' Method." By Seymour J. Sharkey, M.B.

The author's object in writing this paper is to record his success in following out the treatment advocated by Dr. Playfair. Both cases resulted in cure. An excellent account of the method of treatment and the class of cases to which it is applicable is given. The author observes that "too great prominence is given to the nervous system. In the published cases prostration of the nervous system has not been more prominent than prostration of the muscular, glandular, and every other system."

"Cases of Tubercular Meningitis." By John S. Bristowe.

All the cases present particular features of interest, or are specially instructive. (1.) In the first three cases the meningeal affection was obviously secondary, as is usually the case; but the interesting point is that the primary tubercular disease had become retrogressive, and, clinically speaking, a cure had been effected. (2.) In the next three cases the growth of tubercles on the membranes of the brain was apparently slow, and symptoms were developed only when inflammation was superadded, or the growth became so abundant as to involve the surface of the brain itself or the cranial nerves. (3.) Tuberculosis of the membranes, with or without meningitis, is apt to supervene in cases of tubercular tumours of the brain. (4.) In one case the tubercular meningitis was primary, no trace of tubercular disease being found elsewhere in the body. (5.) In one case the symptoms closely resembled delirium tremens. (6.) In conclusion, the author analyses some of the main symptoms occurring in his cases, *e.g.* distension of the ventricles, optic neuritis, paralysis of the cerebral nerves, hemiplegia, convulsions, and temperature.

"Cases of Tubercle of the Cerebellum." By J. S. Bristowe.

The most important facts are:—(1) the existence of the common signs of intracranial tumours, namely, headache, sickness and optic neuritis; (2) the headache was by no means limited to the back of the head; (3) the almost complete absence of anything like a staggering gait, excepting in the last case, in which it had been of long duration and was typical; (4) the supervention in the last case of tubercular meningitis.

"A Case of Spasmodic Wryneck treated by Excision of a portion of the Spinal Accessory Nerve." By Charles A. Ballance.

The patient was a married woman, aged 48, who had suffered severely from spasmodic torticollis for ten years. During the previous ten months the affection had become very severe, and interfered seriously with the taking of nourishment and with sleep. Drugs, electricity, and massage had been tried without avail. The muscles affected were the right sterno-mastoid and the upper portion of the right trapezius. The nerve supplying them, the external branch of the right spinal accessory, was exposed, and about one inch excised, the proximal end at the same time being stretched. The spasm disappeared at once.

Five months later the right sterno-mastoid and trapezius were found to be atrophied. There was some tendency to muscular spasm when she was excited or when the face was directed upwards, but she could by an effort prevent any movement of the head. It was probable that the deep origin of the posterior primary division of the left sub-occipital nerve and also the trapezial fibres of the left spinal accessory were exposed to the same irritation as the deep origin of the right spinal accessory.

The portion of nerve excised was found healthy on microscopical examination.

Mr. Ballance gives an account of the literature of the subject, and also discusses the surgical means to be employed in such cases.

"Clinical Observations on Chorea." By W. B. Hadden, M.D.

The author discusses some of the more unusual symptoms of chorea under five heads, illustrating each group of cases. (1.) Paralysis may be the predominant symptom, preceding the choreic movements, and persisting long after their disappearance. The paralysis often attacks parts which have been little affected by the movements. The paralysis to which attention is called is identical with that occurring in hysteria. In one of the recorded

cases the patellar tendon reflexes were absent. (2.) Complete loss of articulation is illustrated by three cases, in all of which the choreic movements were chiefly right-sided. Speechlessness, like the motor paralysis in chorea, is transitory, both probably depending on inhibition of motor centres, and not necessarily due to exhaustion by antecedent spasm. The seat of the lesion may be in Broca's convolution, or in the co-ordinating centre in the medulla oblongata. (3.) Anæsthesia is not very uncommon in chorea. There seems to be no rule as regards its distribution; although in most of the cases seen by the author the anæsthesia, when unilateral, was limited to the side chiefly affected by the movements, or, when bilateral, was more marked on that side. (4.) Hysteroid convulsive attacks are probably rare, although emotional disturbance is frequent. In the case given the chorea was ushered in by the convulsive attack.

It is pointed out that symptoms of a hysterical nature often supervene in chorea. The author illustrates the converse, the supervention of genuine chorea in hysteria. (5.) Chorea in pregnancy. In most cases there is a previous history of chorea or rheumatism, so that pregnancy can only be looked upon as a possible exciting agent. In one case the tendency to recurrence in succeeding pregnancies is illustrated.

"Epilepsy." By JOHN HARLEY, M.D.

This paper is the outcome of 300 cases of epilepsy observed by the author. Many of his conclusions are corroborative of previous experience, but some are not usually recognised as belonging to so-called simple or idiopathic epilepsy. The following are some of the facts which appear to result from this paper. Epilepsy is essentially a disease of early life. The period of adolescence is more prone to epilepsy than any other. The proneness is greater in females than in males.

During the prime of life (from 20 to 50) the liability is less than half that of the adolescent period. After 50, the liability still more rapidly declines, but in the case of males shows a tendency to rise after the age of 60. Chronic epilepsy is more common in men. The attacks in males are more frequently interrupted by long quiescent intervals, these intervals being much longer in males than in females. Epilepsy may result from imitation or fright. Consciousness may be preserved, partially or completely, during the fit. Consciousness may be suspended, without convulsion. Evanescent insanity, as the immediate con-

sequence of the fit, is common. Disorders of sensation may precede, accompany, or follow the epileptic attack. Disorders of motion may occur, as well as partial paralysis of the tongue and loss of speech. Frequent repetition or violence of the attacks will sometimes produce complete exhaustion of the nervous system and death. (In one of the two cases given a small cysticercus was found in the brain, but there was no sign of irritation or inflammation in the neighbourhood.) Hemiplegia is a frequent result of epilepsy, and even infantile paralysis may result from it.

Onufrowicz on the Auditory Nerve.—Onufrowicz's paper (*Archiv f. Psych.*, xvi. p. 711) is based upon an examination of the brains of two rabbits, whose internal ears had been destroyed on one side as completely as possible by an operation through the outer ear. One rabbit lived six months, the other two and a half months. The brains were subjected to a microscopic examination, and the resulting atrophies noted. The posterior root of the auditory nerve was much atrophied on one side, the anterior root was affected in a considerably less degree. The following are the conclusions of the author:—(1) The so-called "external auditory nucleus," or, as he prefers to call it, Deiters' nucleus, has nothing whatever to do with the auditory nerve. The nuclei were equal in size and healthy on both sides. (2) It is more than doubtful whether the so-called "internal auditory nucleus" has any direct connection with the auditory nerve. (3) The auditory tubercle (Stieda's tuberculum laterale, Stilling's Nacken des Kleinhirnschenkels) is the nucleus of origin of the posterior root of the auditory nerve, that is, of the auditory nerve proper. The auditory fibres of the auditory nerve are in all probability contained only in its posterior root, the anterior root containing the fibres that are distributed to the ampullæ of the semicircular canals. The auditory tubercle is a mass of grey matter that lies to the outer side of the posterior root, and is sometimes described as a portion of the flocculus. It is the homologue of the superior corpora quadrigemina, the centre for the optic nerves, and of the posterior cornua of the cord, the centre for the sensory nerves. The fibres of the posterior auditory root enter the tubercle only after passing through the anterior auditory nucleus, which is the homologue of the spinal ganglia. (4) The place of origin of the anterior root of the auditory nerve is either the vermiform process, or the grey substance of the 4th ventricle underlying the superior cerebellar peduncles, or in both. (5) The

striæ medullares or acusticæ are not direct continuations of the auditory nerve; they are either secondary fibres that issue from the auditory tubercle, or they have, as Longet held, no connection at all with the auditory nerve. (6) Of the further central connections of the auditory nerve we are entirely ignorant. The paper contains a valuable sketch of the views of previous investigators as to the origin of the auditory nerve, and the author emphasizes the superiority of the experimental method of anatomical investigation over even the most careful purely histological researches.

Richter on the Optic Radiations.—Richter (*Archiv f. Psych.* xvi. p. 639) reports the case of a man who, for more than a year, had suffered from homonymous left hemianopsia and other nervous symptoms. The post-mortem revealed on the right side adhesion of the pia mater to the cortex of the three occipital convolutions, the cuneus and the posterior half of the occipito-temporal convolutions, median and lateral; and there was disorganisation of the cortex in these places. There were less marked changes on the inferior surface of the occipito-temporal lobe as far as the tip of the temporal lobe. The white matter underlying the cortex in these regions exhibited fat granule cells, and the tract of degeneration was followed through the centrum ovale along the outer wall of the lateral ventricle into the pulvinar of the optic thalamus. The corpora quadrigemina, corpora geniculata and optic tracts and nerves were quite intact. The author criticises Monakow's statements that occipital lesions are followed by descending degeneration of the anterior corpora quadrigemina, corpora geniculata externa and optic tracts and nerves, and affirms that in man, at all events, occipital lesions have only been proved to cause secondary degeneration of the pulvinar. Another tract of degeneration was traced from the tip of the temporal lobe through the tegmentum to the crusta.

Atrophy of both optic nerves, or even of one, is followed by reduction in the ganglion cells of both optic thalami, all four corpora quadrigemina and all four corpora geniculata, but the author has not observed any change in the occipital cortex.

Westphal on a Peculiar Group of Symptoms.—The patient (*Archiv f. Psych.* xvi. pp. 496 and 778) was a man aged 47, free from syphilitic or alcoholic taint. The first symptoms were paresis of the internal rectus of the left eye, slight double ptosis,

vertigo, and other diffuse cerebral symptoms. Subsequently the lower extremities became weak, and the weakness developed into almost complete paralysis; the upper extremities were affected similarly, but not to such a degree. In the lower extremities various groups of muscles began to display slight degrees of rigidity, the patellar reflexes became heightened, and at a later stage the paradoxical contraction was elicited by movements of the ankle, knee, and hip joints. As the disease progressed, the weakness of the upper extremities increased to absolute paralysis in the case of certain movements; muscular rigidity showed itself, and also the paradoxical contraction. At a later period the muscles of the jaw were affected. The facial muscles showed no marked abnormality.

The motor symptoms were not the only symptoms present, there were striking sensory disorders. Loss of sensibility was first observed in the peripheral portions of the upper and lower extremities, and in the short space of about two years the anæsthesia had extended over almost the entire surface of the trunk and extremities, and ultimately the region of the fifth nerve was affected. The muscular sense was lost, but it is a noteworthy fact that sensibility to heat and cold was always retained, that the superficial reflexes were still evocable, and that the muscles continued to react to electrical stimulation and did not display the usual atrophic degenerative changes. During the whole course of the case, there were no special cerebral symptoms, with the exception of disturbed sleep, vertigo, and feelings of nervous apprehension, and the intelligence of the patient was unimpaired till within a short time of his death. The patient died after an illness of about three years.

The diagnosis of the case was doubtful. It resembled a case of multiple cerebro-spinal sclerosis in some respects—but the widespread anæsthesia, and the low degree of muscular rigidity in the extremities, negatived this view. The post-mortem disclosed disease of the posterior columns of the cord, atrophy of the sensory and motor spinal nerves, and a peculiar change in the tibialis anticus muscle, which was the only one examined; there were a few scattered patches of disease in the lateral columns.

The disease of the posterior columns extended along their whole length, but the root-zones were unaffected, and this probably accounts for the fact that the patellar reflex was retained. The absence of ataxia, notwithstanding the extensive disease of the posterior columns, is attributed to the slight intensity of the

disease, and to death occurring before its full development; it may also perhaps be due to the peculiar localisation of the disease, for we do not yet know what portion of the posterior columns have to be diseased to produce ataxia. The sensory symptoms are explained by the affection of the peripheral nerves, which can be considered either as a primary atrophy or as a chronic parenchymatous neuritis, and which probably had its origin in the distal terminations of the nerves. The peculiar changes in the muscles, Westphal thinks, may be related to the paradoxical contraction.

Rumpf on Syphilis, Infantile Paralysis, and Cerebellar Atrophy.—*Syphilis of Nervous System.*—A man, aged 31 (*Archiv f. Psych.* xvi. p. 410), contracted syphilis when 29, and subsequently suffered from a cutaneous eruption and sore throat, and from a suddenly developed attack of right hemiplegia of the limbs. Symptoms of spastic paralysis of the left leg next showed themselves, then incontinence of urine and faeces. The patellar reflexes were exaggerated on both sides, a slight girdle sensation was present, and a few months before death sensibility became impaired in both lower limbs. The patient died at the age of 32. The chief post-mortem results were:—(1) An old lesion in the left internal capsule, with secondary degeneration of the pyramidal tracts of the right lateral column of the cord; (2) degeneration of the left lateral column, chiefly of its pyramidal tracts, in the cervical region; (3) degeneration of Goll's columns in the cervical regions; (4) a patch of inflammation in the dorsal portion of the cord, implicating the lateral and posterior columns of both sides, and gradually increasing in dimensions till the lumbar region was approached, where the disease again contracts its limits, and is confined to the posterior columns, and to the postero-lateral parts of the lateral columns; (5) the blood-vessels throughout the cord, but especially in its dorsal region, presented proliferative changes in their three coats, the inner coat being affected later than the outer coats—these changes are often found associated with syphilis, but they are not pathognomonic of it, being found in other diseases.

Infantile Paralysis.—A case of infantile paralysis (*polio-myelitis anterior acuta*) is described in which there was atrophy of the central convolutions of the cerebrum. The paralysis affected the right side, and came on in the fourth year. Death occurred from pneumophthisis in the eighteenth year. The middle and lower parts of the central convolutions of the left cerebral hemisphere were

smaller than the corresponding parts of the right. The author states that the dimensions of the left internal capsule (in front of the genu) and of the left lenticular nucleus were markedly less than those of the corresponding structures on the right side. He regards the cerebral atrophy as an arrested development, the result of the spinal paralysis.

Cerebellar Atrophy.—The paper closes with the case of a cat which had symptoms of cerebellar disease, and which was found after death to have atrophy of the cerebellum.

Schulz on Aphasia, Hemianopsia, Spinal Sarcoma, and Tetanus.—Schulz (*Archiv f. Psych.* xvi. p. 579) reports the following four cases:—

Aphasia.—A man aged 20 suddenly fell from his cart on March 21 without losing consciousness. On March 24 his speech became affected, and occasional convulsive movements were noticed in his right face. On March 29 he was admitted into Hospital suffering from complete amnesic aphasia, nevertheless he understood all that was said to him, and could repeat words that were uttered slowly to him. There was paresis of the lower half of the right face, the tongue deviated to the right, and every three or four minutes there were strong convulsive twitchings in the whole of the right face, with temporary loss of consciousness. The aphasia became absolute, the convulsions continued to grow worse, and ultimately became general, and the patient passed into a comatose condition and died on March 31. The autopsy disclosed a cheesy tumour, that was probably tubercular and was of the size of a hazel nut, in the inferior frontal convolution of the left hemisphere (Broca's convolution).

Hemianopsia.—A woman aged 49 had temporal hemianopsia of the left eye (18 months before, she had suddenly felt something come over her left eye), a reeling gait, occasional vertigo, and right hemiparesis; and her mind was childish. Under iodide of potassium treatment she improved, but in two months it was noticed that there was complete left homonymous hemianopsia, the right eye being now affected. Six months afterwards, on April 1, 1884, the patient was admitted into hospital much worse; the hemianopsia remained; there was paresis of all four extremities, which gradually developed into almost complete paraplegia, with contracture of the limbs, heightened tendon reflex and sensory disturbances. At a later stage there was paralysis of the sphincters and extensive bedsores, and the patient died on Sept. 3, 1884.

During the last three months she was demented. The diagnosis was:—Tumour springing from the hypophysis cerebri compressing the right optic tract, and latterly implicating the pons. The autopsy, however, showed chronic cerebral pachy- and leptomeningitis, interstitial encephalitis, and hypertrophic cervical pachymeningitis at the level of the 3rd and 4th cervical vertebræ. The pia mater was especially thick and adherent in the region of the chiasma, and to this Schulz is inclined to attribute the hemianopsia, though he admits the evidence is not at all conclusive. The spinal cord was softened opposite the 3rd and 4th cervical vertebræ.

Spinal Sarcoma.—A girl of sixteen had complained for three months of pain in the back, and during the last three weeks had been troubled with numb feelings in the hands and arms, and her arms had gradually become paralysed. On admission into hospital on March 31, 1881, there was almost complete paralysis of both upper and lower limbs; loss of sensibility over the whole of the upper limbs, and vaso-motor disturbances evidenced by hot, red, and swollen hands; diminished sensibility in the lower limbs, with partial loss of the deep and superficial reflexes, and paresis of the bladder. Acute pains were still felt in the back, and soon respiration became affected, and the patient died on April 2, 1881. The diagnosis was acute meningo-myelitis. At the post-mortem a tumour was found ensheathing the cord from the cauda equina upwards to the upper cervical region. It had the characters of an alveolar sarcoma, and had evidently originated in the pia mater. In some places, notably in the upper dorsal part of the cord, and the upper cervical part, what little of the cord was left, was completely softened.

Tetanus.—The author reports a mild case of tetanus in which the irritability of the facial nerves to mechanical stimulation was increased, but their irritability to the stimuli of the constant current was normal. This last observation agrees with Erb's statements, but it is in opposition to what other authorities have found in this disease.

W. J. Dodds, M.D., D.Sc.

Report of a slight Outbreak of Epidemic Cerebro-spinal Meningitis, with Notes of Six Cases. By Wm. Frew, M.B., C.M.—This outbreak occurred in the town of Galston, near Kilmarnock. It originated, the author thinks, through the bad

sanitary conditions of the place; there was an accumulation of the sewage in the neighbourhood of the houses. Personal intercourse, however, could be traced from the first patient down to the last (with one *possible* exception).

Case I.—Initial symptoms, headache and vomiting: on second day, temperature 104°, followed by delirium, sordes on lips, &c. By fourth day, improvement set in. No rash was observed. After recovery, absolute deafness, staggering gait. Girl, age 10.

Case II.—Æt. 1. Vomiting and pain. Temperature 104°. Measly rash on forearms and limbs. Death within fourteen hours of onset.

Case III.—Sister of Case II. Age 7 years. Rigor, headache, abdominal pain. Next day, vomiting. Third day, temperature 103°–104°, much restlessness and pain in head. Fourth day, worse; photophobia, feeling of constriction at throat, mottled purplish rash on forearms and legs; legs flexed on abdomen, retraction of head; delirium, &c., towards night. Sixth day, improvement, tenderness along spine. Recovery tedious, relapses of pain, vomiting, &c., much exhaustion; but no paralysis or affection of special senses.

Case IV.—Girl, 8½ years old. A very acute case; chief symptom, vomiting; death in a single night. *Post-mortem*, petechial spots on legs: hyperæmia of membranes, with inflammatory exudation beneath them, at vortex of brain, over cerebellum, and along spinal cord.

This girl had visited Case III. three days before her own seizure.

Case V.—Vomiting, fever, convulsions affecting principally the left arm and left angle of mouth. No rash. Death on fourth day.

Post-mortem.—Calvaria of a bright pinkish colour: venous congestion of surface of brain; gelatino-purulent exudation on upper and under surface of brain (especially right anterior part), and also over cerebellum and spinal cord.

Case VI.—Age 14 months. Vomiting, fever, convulsions, mouth and eyes drawn to right. Examination of the fundus oculi showed no neuritis, but distinct hyperæmia. No rash observed. Death on fourth day.

On the Pathology of Pseudo-hypertrophic Muscular Paralysis, with Remarks on a so-called Degeneration of the Nervous System. By G. S. Middleton, M.A., M.D.—Two cases

were examined—in the first case the microscopic changes were as follows:—

Muscular system.—The muscular fibres varied greatly in thickness; some muscles (and especially those which were otherwise least abnormal) were in a condition of hyaline degeneration; in others there was increase of connective tissue between the fibres, or infiltration with fat. In the heart and bladder there was increase of interstitial connective tissue, but no fatty infiltration, nor hyaline degeneration.

Nervous system.—The only change found was the presence of small whitish glancing bodies, scattered in large numbers, and indifferently (though more numerous in the white substance than in the grey) through all parts of the nervous system that were examined. They stained in logwood, and faintly with osmic acid. These the author justly concludes to have been produced by the method of preparation, the parts having been hardened in alcohol.

In Case II. the muscular tissue presented a marked fatty infiltration, some increase of connective tissue, and atrophy of muscular fibre. No hyaline degeneration. Nervous system practically normal.

From a consideration of these cases, and a review of the literature of the subject, the author concludes that the disease is essentially a myopathy.

Beevor on Three Cases illustrating the Localisation of Motor Centres in the Brachial Enlargement of the Spinal Cord (*Roy. Med. Chir. Soc.*, April 14, 1885).—These cases illustrated the groups of muscles found by Ferrier and Yeo (*Proc. Roy. Soc.* No. 212, 1881), to be thrown into action when each motor root in the monkey's brachial plexus was faradized. In the first case—progressive muscular atrophy—in the fourth cervical group the deltoid, rhomboid, biceps, brachialis anticus, supinator longus, extensors of fingers were severely affected on both sides, and in addition on the left the supra- and infra-spinati; the fifth cervical (clavicular part of deltoid, biceps, brachial. antic., supin. long. serrat. mag., extensors of fingers) was completely paralysed; in the sixth cervical (lat. dorsi, pect. maj., triceps, serrat. mag., pronators of wrist), only the two latter, and the last only slightly; in the seventh cervical (lat. dorsi, subscap., pect. maj., triceps, teres major, flexors of fingers), the teres was affected on both sides, and the pect. maj. slightly on the left and the flexors of the fingers on the right; in the eighth cervical (long flexors of wrist, ulnar

flexors of wrist, small muscles of hand, extensors of fingers, triceps (long head), pect. maj.), all the muscles were severely affected on the right, except the last two, and extensors of fingers and pectoralis maj. slightly paralysed on the left; the first dorsal (small muscles of hand and interossei), on right only. The above shows the biceps type, viz. fourth and fifth cervical on both the right and left, besides the eighth cervical and first dorsal on the right. No reaction to faradic current was obtained in the severely affected muscles.

The two other cases—triceps type—occurred in boys of 18 and 11 years old, who had infantile paralysis when young.

In one case on the right side, the only muscles affected were the triceps, latissimus dorsi, pectoralis major (sternal part only); these were absolutely powerless, and the last muscle had completely disappeared; they gave no reaction to faradic or constant currents; they belong to the sixth and seventh cervical groups. On the left side, beside these muscles, all muscles corresponding to the seventh cervical, except flexor digit., and all those of the sixth cervical, except pronators of wrist, were paralysed; in the fifth cervical group, the deltoid, serrat. mag., extensors of wrist were only slightly affected; and in the fourth cervical, only the rhomboid, supra- and infra-spinati, teres minor were severely affected: there was no reaction to the constant current in the severely paralysed muscles.

In the other triceps case, the teres major, latiss. dorsi, subscap., pect. major (lower half), triceps, serratus magnus and the rhomboid (?), were the only muscles paralysed, and these, excepting the two last, are supplied entirely by the sixth and seventh cervical roots, so that the muscles corresponding to the biceps type have escaped.

It should be mentioned that Ferrier has since found (*Proc. Roy. Soc.*, No. 225, 1883) that his original list, used in this paper, is one root too high, and the groups of muscles should refer from the fifth cervical to the second dorsal, instead of from the fourth cervical to the first dorsal, so that the group here assigned to the fourth belongs to the fifth, &c.

Case of Tubercular Tumour of the Cerebellum. By William Vost, M.B., C.M. (*Glasgow Medical Journal*, July 1884.)—Onset of symptoms ten days before death; purpuric rash, cough malaise; then staggering gait: pain left side. On admission, could not walk, lay with knees flexed, pain on extending them. Pupils

large, not responding to light; eyes fixed, but slight to and fro movements of them later on. Tâche cérébrale; bladder partly paralysed. Semi-comatose condition. Temperature 100°–102·4°. *Post-mortem*, phthisis, with old pleurisy of left lung; and a tumour (tubercular) on the under-surface of the left lateral hemisphere of the cerebellum.

J. A. Ormerod, M.D.

Remark on a Case of Bilateral Paralysis of the Spinal Accessory Nerve (*Deutsche Med. Wochenschrift*, No. 27, 1885. Case exhibited at Verein für innere Medicin, June 1885).—The patient, a man, aged 35 years, contracted indubitable syphilis in 1873. Six months before being exhibited to the Society he began to suffer from pains in the neck and right shoulder, with inability to raise the shoulders; six weeks subsequently his voice became husky.

He presented the following state:—Over the sternal end of the right clavicle an exostosis as large as a hen's egg, and a similar but smaller exostosis over the sternal end of the left clavicle; hard prominences from the right side of the upper cervical vertebræ, and from the left side of the sixth and seventh cervical vertebræ; marked atrophy of the trapezii, levatores anguli scapulæ, rhomboides, muscles of the back of the neck and of the pectorales; the sterno-cleidomastoides only slightly wasted, and the extrinsic muscles of the larynx healthy; both deltoides fairly developed, but the muscles on the flexor aspect of the upper arm atrophied, especially on the left side, on which the supinator longus is similarly affected; muscles on the extensor side of both forearms only rather flaccid; those on the flexor side and muscles of hands intact. Motor paralyses exactly corresponding to the distribution and degree of the atrophy easily demonstrated; characteristic separation of the inner border of the scapula from the chest wall when the arm is raised, shows the left serratus magnus to be also involved. Respirations 24–27 per minute, without stridor or dyspnœa, thoracic in type, with respiratory epigastric recession (diaphragmatic paralysis); pulse-rate, 90–115 per minute; acceleration possibly secondary to hurried respiration only; occasional fibrillar twitching of sterno-cleidomastoideus, deltoideus, pectoralis, biceps, and supinator longus on both sides.

On electrical examination, platysmata, muscles of the root of the tongue and extrinsic muscles of the larynx react normally; sterno-mastoides react to moderately powerful faradic current;

but trapezii and left biceps excitable only by very strong current, and their contraction slow and incomplete (faradic degenerative reaction). Galvanic degenerative reaction marked in trapezii, sterno-mastoides, left biceps, and supinator longus; present, but to a less degree, in deltoides; in flexors of arm on right side, and extensors of wrist on left side.

The hoarseness of voice suggested the probability of the intrinsic laryngeal muscles, supplied by the inner division of the spinal accessory nerve, being affected. On April 1st, Dr. Böcker observed the signs of paralysis of the left recurrent laryngeal nerve; on May 6th the right vocal chord appeared the more immobile; on May 16th both chords occupied a position intermediate between the cadaveric and that characteristic of paralysis of the crico-arytænoides postici. On phonation the chords do not approximate, and vibrate coarsely, attributable to paralysis of the crico-thyroides, supplied by the external laryngeal nerve.

There are no signs or symptoms of implication of any of the cranial nerves, except slight difficulty in swallowing, explicable by involvement of the pharyngeal filaments of the spinal accessory (Burchard).

The author considers a syphilitic process—probably a pachymeningitis, extending laterally as far as the ligamentum denticulatum, and involving the anterior roots of the upper cervical nerves, and of the spinal accessory as far down as the fifth and sixth nerves—the most likely explanation of the phenomena observed.

The interest of the case culminates in the laryngeal condition, which the author considers as a fact opposed to the well-known views of Semon regarding the special liability of the abductor fibres to become paralysed in disease affecting the nucleus of the spinal accessory or its peripheral branches. Such an argument appears hardly sound. The chords seem to have occupied a position exactly corresponding to partial abductor paralysis, as has been elsewhere pointed out by B. Fraenkel.

Hughes Bennett on Locomotor Ataxy, without Disease of the Posterior Columns of the Spinal Cord. (Vol. xviii. *Clinical Society's Transactions*, 1885.)—Dr. Bennett gives the details of a case which appears to us to be unique from many points of view. It is in itself not only of great clinical interest, but the conclusions which the author draws from the facts are in

our opinion calculated to throw an important light on the physiology and pathology of so-called tabes dorsalis.

A gamekeeper, 48 years old, having previously been in good health, and never having suffered from syphilis or other illness, about nine months before he came under observation gradually experienced attacks of shooting-pains in his legs, and inability to walk properly. This was accompanied with a sensation of numbness in the limbs. All these symptoms slowly increased in severity. On examination, the general health was found good, and all the functions and organs of the body normal, with the following exceptions:

There were occasional slight attacks of giddiness, and slight nystagmus, otherwise the cerebral functions and special senses were normal. The movements of the upper limbs were somewhat weak, and slightly but distinctly ataxic. The bladder, rectum, and sexual organs appeared to be normal. The lower limbs presented all the typical characters of ataxy of an advanced type, the incoördination being markedly increased in the dark, or with the eyes closed. The plantar reflexes were diminished, and the knee-jerk on both sides was totally abolished. In the feet and legs the cutaneous sensibility was slightly diminished and retarded; otherwise and elsewhere it was normal. There were frequent attacks of severe lancinating pains in the lower limbs. The nutrition of the muscles in their electrical reactions was normal. During a residence of two and a-half months in the hospital all these symptoms somewhat rapidly augmented, and the patient died of exhaustion.

On post-mortem examination the essential lesion was found to consist of a series of isolated sarcomatous tumours, situated chiefly in the posterior portion of the pia mater of the cord. These involved the posterior spinal roots, without causing their serious degeneration. These tumours were most numerous in the lower dorsal and lumbar regions. The posterior columns of the cord were everywhere perfectly normal. The medulla oblongata to the naked eye appeared natural, but the microscope revealed a sarcomatous growth occupying the floor of the fourth ventricle, and extending downwards to the centre of the wall surrounding the central canal.

From these facts, of which only a superficial sketch is here given, the author draws important pathological and clinical conclusions. The point to which special attention is directed is this: while during life the patient presented most of the charac-

teristic phenomena of tabes dorsalis, the posterior columns of the cord were found after death to be everywhere normal in structure, while, on the other hand, the posterior spinal roots alone were extensively involved in the morbid lesion. Thus evidence is adduced to show that, for the production of those groups of symptoms comprised under the term of tabes dorsalis, disease of the posterior columns of the cord is not an essential factor, and that they may be fairly developed, the centre being in a perfectly normal condition. The case supports the view, that incoördinate movements may be produced by interruption of the afferent elements of the nervous system. It moreover forms an important pathological link between the classical lesion in the posterior root-zones, and the cases of "nervo-tabes" périphérique of Déjérine.

Here we can do no more than merely hint at the leading points of this valuable observation, and must refer our readers to the original paper for the facts in detail, as well as for a close and scientific reasoning thereon by the author. A series of beautifully executed lithographs of the natural eye and microscopical appearances are appended to the communication.

Hughes Bennett on a Case of Cerebral Tumour, the Surgical Treatment by Rickman J. Godlee. (Vol. lxviii., *Med. Chirurg. Trans.* 1885.)—The incomplete reports of this case which appeared in the different medical journals at the time of its occurrence excited a considerable amount of interest in the profession. In the *Medico-Chirurgical Transactions* for 1885 appears the first authoritative statement from the medical men in charge of the case. This consists of a full and complete record of all the circumstances connected with what we believe to be still a unique case. It will be unnecessary to abstract them in detail, as the general results are now well known. Those interested in the subject will do well to consult the original paper, which is carefully drawn up and fully illustrated. The history and condition on examination of the patient prior to treatment is described, the reasons for the diagnosis discussed, and the necessity for heroic treatment defended. The various steps taken to remove the tumour from the substance of the brain are detailed, and all the phenomena succeeding the operation are recorded. The post-mortem appearances are described, and the condition of the cerebral cortex demonstrated by means of engravings.

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Finally, a commentary on all circumstances of the case is given, dealing chiefly with the question of diagnosis, the surgical operation, the clinical phenomena following the operation, and the revelations of the autopsy. In the course of the discussion many interesting facts are demonstrated, and suggestive ideas advanced, on the question of cerebral tumour and the possibility of removing it by surgical operation. The authors conclude by saying that, "Although unfortunately in this instance life was not permanently preserved, the experience we have gained by this case leads us to the belief, that there is an encouraging prospect for the future of cerebral medicine and surgery; and that, as a tumour of the brain can be diagnosed with precision, and successfully removed without immediate danger to life, we confidently anticipate that under more favourable circumstances the operation will be performed with lasting benefit to the patient." Their previsions, as the reader is aware, have been justified by the results recently obtained by Horsley and others; and the surgical treatment of cerebral tumours situated in the motor region has been proved in several cases to be both safe and effectual.

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END OF VOL. IX.

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